LIBRARY
BOSTON UNIVERSITY
SCHOOL OF MEDICINE

The Journal of
Thoracic Surgery

VOL. 7 OCTOBER, 1937 No. 1

Original Communications

BLOCKED CAVITIES IN PULMONARY TUBERCULOSIS*

LEO ELOESSER, M.D.
SAN FRANCISCO, CALIF.

THE American Association for Thoracic Surgery has helped not a little toward a clearer insight into the processes of pulmonary disease. We may be proud of the work of many of our members (Adams and Singer, Alexander, van Allen, Coryllos, Graham, Chevalier Jackson, to mention a few), who have taken an active part in clarifying pulmonary pathology by clinical observation and experiment. Studies of the mechanics of respiration and of changes in the bronchial tree have succeeded in explaining obscure pictures of disease and have made us modify or reverse various inadequate notions. Older concepts of cavity formation in pulmonary tuberculosis, as a process during which a tuberculous infiltrate breaks down and is discharged through the bronchi, were insufficient in many directions. They failed to explain why some cavities were branching and multilocular, while others were smooth and round; they failed to explain why occasionally a small infiltrate in a not very toxic patient would break down into a huge round cavity and why, again, a large massive density might disappear without a sign of excavation. Various more or less unsatisfactory and complicated theories that concerned changes in retractility of the diseased lung were called upon to explain the shape, growth, and distribution of tuberculous cavities. It is scarcely worth while to review them all; Adams and Singer,[1] Coryllos,[2] and Graef[3] have already done so. There has long been a suspicion that other processes besides mere caseation and discharge of a soft focus through the bronchial tree were responsible for certain forms of cavitation and that these processes were to be sought in localized changes in the bronchi.

Presidential Address read before the Twentieth Annual Meeting of the American Association for Thoracic Surgery, at Saranac Lake, N. Y., May 31, June 1, 2, 1937.

*From Stanford University Surgical Service and Tuberculosis Division, San Francisco Hospital.

Recent interesting experiments of Moolten[4] have laid a basis for a purely mechanical explanation. Using human and animal cadaveric lungs, he was able to produce both spherical and irregular cavities, in a purely mechanical way, by biting off portions of the bronchial wall and the interlobar septa with punch forceps. Before inflation of these lungs, the injuries took the form of simple lacerations or elongated tears; after inflating them or subjecting them to negative pressure, spherical cavities were formed which resembled natural ones roentgenographically and on section. There was of course no question of disease playing a rôle in the formation of these cavities. Their size was determined by the amount of pressure used for inflation rather than by the extent of injury. Some cavities refused to deflate even when suction was made on the lung; these were kept inflated by a valvular flap of torn tissue, which sealed off their bronchial connection. Reinders[5] thought that retractility of the neighboring lung pulled the cavity out into a sphere. He illustrated this theory with a simple mechanical model, consisting of a piece of sheet rubber, into which he burned holes of various shapes. When the rubber was distended, the irregular hole assumed a round form; if a further portion of the edge of the defect was seared, the defect did not become irregular or clover-leaf in shape but remained round and merely increased in size.

These experiments demonstrate the fact that (1) if the more or less unyielding framework that anchors the resilient surrounding mass of the lung to its root is interrupted; (2) if the resilient lung is subjected either to internal air pressure or external suction (the pull of the chest wall); and (3) if air is allowed to enter the site of rupture; a spherical cavity results. Thus a pulmonary cavity may be formed by a combination of three mechanical factors alone, viz: (1) interruption of the unyielding pulmonary framework; (2) internal pressure or external suction on the lung; (3) access of air to the site of injury.

Perhaps the combination of these three factors determines purely mechanically the shape and development of the spherical pulmonary cavity which documents itself in the x-ray as an annular shadow. If the links in this chain are not complete, a typical globular cavity does not result; caseous ulceration of the lung then gives rise to shaggy-walled, irregular abscesses, such as one sees in tuberculous abscess of the kidney, for instance, or of the lymph nodes or other solid organs without rhythmical distensile forces such as act upon the lung.

A step further—it is easier for air to enter the bronchial tree than it is for it to escape from it; inspiration is the short phase, expiration the long one of the respiratory cycle; the bronchi are wider during inspiration and narrower during expiration (Heinbecker and others);[6] simple bronchial stenosis tends to entrap inspired air distal to the obstruction; all of these factors are greatly increased by irregularities in the bronchial

wall, or by sticky secretions in the lumen, which tend to increase difficulties of expiration and which affect inspiration comparatively little, so that most bronchial inflammations and ulcerations tend toward a stop valve effect (C. Jackson and C. L. Jackson).[7]

It is easy to see, therefore, how destructive processes originating in the bronchial wall, or breaking through into it and ulcerating it secondarily, will cause pulmonary excavation if they bring about a conjunction of the factors noted above, viz: destruction of the framework, maintenance of respiratory movement, and access of air to the injured area. It is furthermore easy to see how persistence of these factors will, by increasing pressure in the pulmonary cavity, keep the cavity distended in a globular shape, and once the conditions for its formation are given, cause rapid increase in its size.

If this is true one should expect to find that certain fresh pulmonary cavities, situated in a lung that is still more or less resilient and not too fibrotic nor too much carnefied, have their bronchial openings more or less continuously and more or less completely blocked. One should expect that such cavities should contain air under higher than intrapleural pressure. It is not necessary, of course, that this cavity pressure be explosive; it need only be constantly higher than the remaining intrathoracic pressure in order that the cavity be kept distended. It need not even be constantly positive, for one should expect that at the height of inspiration the chest wall might still exert a pull on the distended cavity, which would bring its pressure below that of the atmospheric air. Furthermore, if the cavity is large, fresh, and thin walled, one should expect to find respiratory pressure variation communicated through its thin wall to its interior and one should expect to find pressure in the cavity lower on inspiration and higher on expiration, even though the bronchial communication be completely blocked.

The sudden ballooning that takes place in congenital cysts of the lung is typical of this mechanism. Infants in whom this occurs present the clinical picture of a pressure pneumothorax with intense dyspnea, ballooning of the affected side, and contralateral displacement of the mediastinum. Robert T. Miller, Jr.'s, case is a typical example.[8] Figs. 1 and 1*A* represent a patient seen at the First Surgical Clinic of Moscow University (Prof. Burdenko). The man of twenty-seven years of age had a thin, round annular shadow in the left chest which had been discovered years before. It remained symptomless and had been observed on repeated x-ray examination. Suddenly, after an acute respiratory infection, the man became alarmingly dyspneic and cyanotic; the heart and mediastinum were displaced into the right chest, and the annular shadow was much increased in size, filling almost the entire left side of the thorax. At operation the lung was found to contain a cyst, containing air and pus under much pressure.

These ideas that changes in the bronchi may be responsible for certain kinds of excavation and for conditions obtaining in certain cavities are not new. It is true that standard texts on pathologic anatomy and tuberculosis still cling to the sequence of tuberculous infiltration, caseation, liquefaction, and rupture into the bronchi as an explanation of cavitation, and that the mechanical factors resulting from respiratory movement of air in the bronchi have not been precisely formulated and clarified, but scattered observers and experimenters have accumulated facts expressive of their dissatisfaction with older concepts. Moolten's recent experiments, demonstrating the possibility of a purely mechanical production of cavities in the cadaveric lung, are of basic importance. Salkin, Cadden, and McIndoe[9] filled the bronchial trees of a series of tuberculous cadavers with an opaque mixture; in some of the cadavers

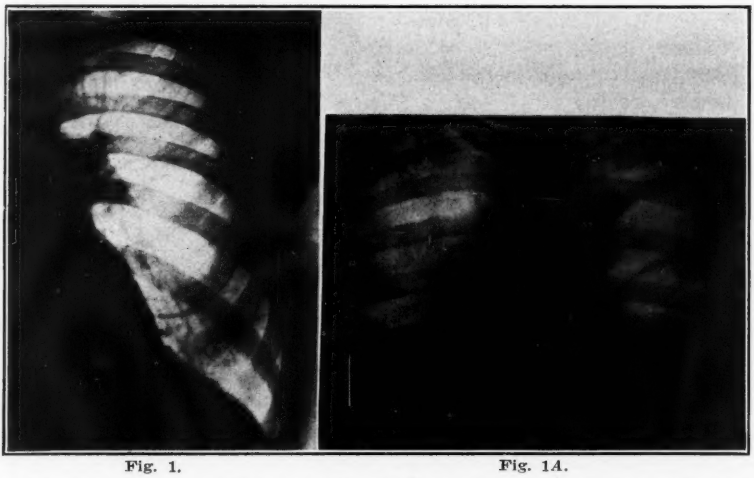


Fig. 1. Fig. 1A.

Fig. 1.—A large congenital cyst of the left lung, seen as a perfectly round, thin-walled annular shadow, remaining stationary for years.

Fig. 1A.—The cyst ballooned so that it fills almost the entire left chest, displacing the heart into the right chest, causing intense dyspnea and cyanosis.

tuberculous cavities were filled with opaque fluid by needling them directly through the chest wall. Their studies showed that of 70 tuberculous cadavers, 18, or 26 per cent, had blocked cavities; and that of 147 cavities over 5 cm. in diameter, 21, or 14 per cent, had no demonstrable connection with the bronchi. They reproduce x-ray films taken during the life of some of the subjects of their studies, which show cavities alternately closed and open, represented by uniform densities and air-containing spaces with a fluid level, respectively. They think that the presence of air and fluid in these cavities is explained by a theory that air and fluid may pass through a cavity wall in either direction.

Various clinicians have occupied themselves with the pathogenesis of excavation. Hall[10] was one of the first to call attention to ballooning

of the lung in pulmonary tuberculosis. He describes a cavity simulating pneumothorax in which valvular action of a damaged bronchus permitted air to enter, but not to leave, the cavity; and he mentions ballooning of the lung, which he considers to be caused by a "semidetached piece of lung blocking the bronchus, permitting air to enter, but for the time being not allowing any to escape." Blyth-Brooke[11] needled an inflated cavity and found a pressure of +4 +6; after withdrawing 100 c.c. of air, the pressure fell to −2 +2; withdrawing another 100 c.c. produced no change, so that Brooke considered that he was dealing with a valve-like obstruction. Pearson[12] in a paper on the "Pathogenesis of Pulmonary Cavities" considered that many cavities were due to inflation. He needled a large cavity through the chest wall and found manometric pressure readings of +4 +16; after withdrawing 250 c.c. of air, the pressure read 0 and +11; after 500 c.c., 0 and +9. Morland[13] thinks that cavities are formed by a combination of destruction and inflation. Sanes and Kenny[14] describe a huge tension cavity in a tuberculous infant; Korol[15] described one which expanded when the communicating bronchus was plugged with a blood clot. Most prominently we will recall the work of Coryllos[2] and his pupils,[16] who have occupied themselves extensively with the investigation of cavities and closure of the bronchial opening of a cavity as a means of cure.

The relation of pulmonary cavities to the bronchial tree is important not only theoretically but clinically. If a partially or intermittently blocked bronchus can keep a cavity inflated and prevent its cure; if more permanent and total bronchial occlusion can cure the cavity, as Coryllos believes, or on the contrary cause it to increase in size and make more toxic symptoms, as most others believe, then accurate anatomic diagnosis of the bronchial relations of a cavity should be important for the patient's welfare.

DIAGNOSIS

X-ray.—Accurate anatomic diagnosis may be difficult. That there is some agent, other than the pull of the chest wall, which keeps certain cavities inflated, becomes apparent in studying films of some cavities which refuse to collapse in spite of what is apparently a completely free pneumothorax. Occasionally one sees a pneumothorax which seems to be free of pleuritic adhesions, but into which certain upper lobe cavities stand out like a bubble from the top of an otherwise deflated lung (Fig. 5). At times there are adhesions, but severing them seems to make no difference in the size of the cavity. Again, severing adhesions may cause the cavity to shift its position in the chest, although its size remains unchanged. A similar shift may take place after thoracoplasty, or even spontaneously, as one or the other part of a lobe in which the cavity lies shrinks or expands with varying degrees of bronchial occlusion and varying air content (Figs. 17, 18, 30, 31). Fischel[17] has called

attention to such "wandering" cavities. If a cavity grows larger after induction of pneumothorax rather than smaller, it may be regarded as blocked by a stop-valve.

CASE 1.—A nineteen-year-old, part Indian boy, both of whose parents had died of tuberculosis, was diagnosed in 1930, at the age of thirteen, as having "childhood tuberculosis." In the fall of 1935 he complained of weakness; a film in January, 1936, evidenced a cavity. Pneumothorax was begun. Expectoration was minimal; what little sputum could be examined contained tubercle bacilli. A thin-walled, uncollapsed cavity remained at the right apex. Closed pneumolysis was done twice on June 13 and July 1, 1936. Several bands were severed, but the cavity remained unchanged. On July 17, 1936, the chest was opened and a large broad adhesion was ligated and severed. The cavity still remained unchanged. On Sept. 26, 1936, a thoracoplasty was done (first to fifth ribs); during operation the cavity was needed and lipiodol injected into it. On Oct. 12 the cavity was again needled; pus withdrawn from it contained myriads of tubercle bacilli; lipiodol and methylene blue were injected into it. A note made two days later states that neither lipiodol nor methylene blue appeared in the sputum. On Dec. 5, 1936, thoracoplasty was continued, the sixth and seventh ribs being resected. The patient still expectorates very little, but his weight has decreased and he has steadily lost ground. The cavity remains unchanged.

Here then is a cavity which does not collapse, although it lies free in a pneumothorax. Bacillus-laden pus is aspirated, lipiodol and methylene blue are injected, but the patient expectorates neither pus nor lipiodol nor methylene blue. It is unlikely that the cavity communicates with a bronchus at all; it resists all kinds of collapsing measures and does not heal.

A cavity which does not wink under the screen is suspicious of not having an open bronchial connection; if it dilates when the patient coughs, instead of growing smaller, it is likely that the bronchus leading to it closes by a stop-valve mechanism. Such variations in size may be difficult to make out accurately, for with stenosis of the larger bronchi the mediastinum moves toward the stenotic side on coughing and this movement may make movements of the cavity wall difficult to interpret.

NEEDLING THE CAVITY

Needling the cavity may yield valuable information. Needling, of course, is permissible only when one is sure that the pleural space is obliterated. It should be done under the screen and provision should be made for taking immediate films without moving the patient from the table (fluorographic equipment if possible). One may withdraw pus from the cavity; if there is little or no expectoration, or if the pus from the cavity contains acid-fast bacilli and the sputum consistently does not, the cavity may be regarded as blocked. If the needle strikes air, the cavity should be watched under the screen while the plunger of a 10 c.c. syringe is moved back and forth. The patient should previously have been trained to hold the glottis open and stop breathing. When he does this, alternate pressure and suction on the syringe should, if the cavity is blocked, cause corresponding movements of the cavity wall.

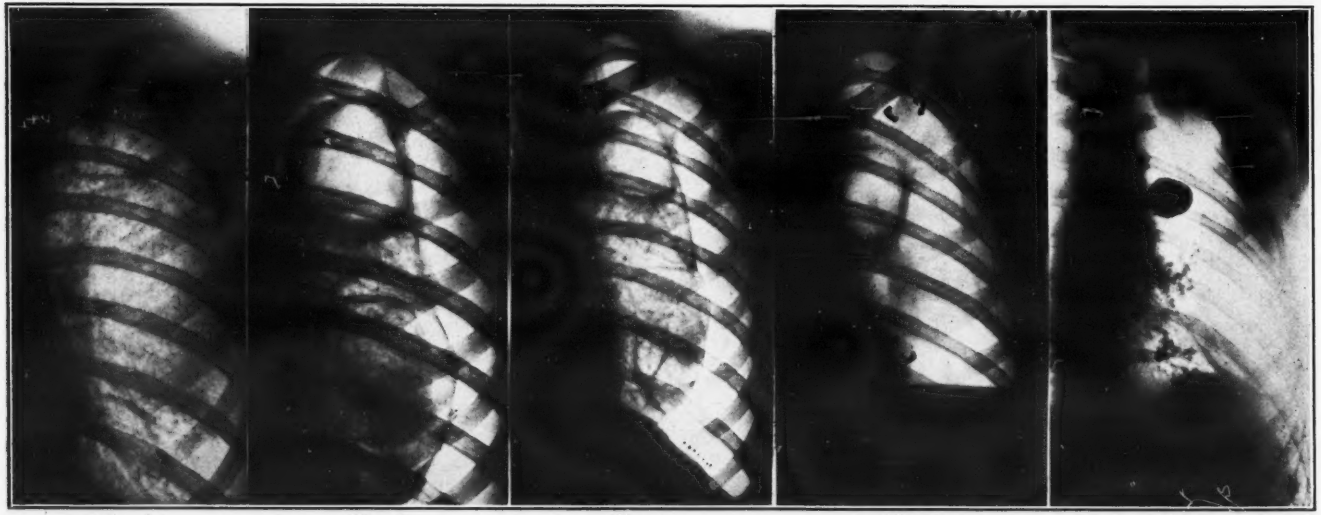


Fig. 2. Fig. 3. Fig. 4. Fig. 5. Fig. 6.

Fig. 2.—Case 1. Large, thin-walled, annular shadow at right apex.

Fig. 3.—Case 1. June 23, 1936: after pneumothorax; cavity unchanged in size.

Fig. 4.—Case 1. July 13, 1936: after closed pneumolysis; cavity unchanged.

Fig. 5.— Case 1. July 23, 1936: after open pneumolysis; cavity unchanged.

Fig. 6.—Case 1. Sept. 28, 1936; after thoracoplasty; cavity injected through the chest wall with lipiodol (the alveolar lipiodol was injected perorally at a previous sitting).

Manometric pressure in the cavity should be measured. To do this is not quite easy. One should make sure by repeated fluoroscopic control that the point of the needle has not deviated from the cavity. The syringe should be detached from the needle and the manometer attached quickly. If there is delay in attaching the manometer to the needle, sufficient air may escape to lower the pressure in a small cavity considerably. Constant pressure without respiratory oscillation indicates a thick-walled cavity; high pressure, a stop-valve block. However, the pressure need not be actually high to indicate a block; it need merely be higher than that in the pleural cavity to indicate some impairment of bronchial communication. What the intrapleural pressure is we have no means of knowing, for it is not legitimate to needle a cavity through a free pleura. Neither do respiratory fluctuations of the manometer refute a diagnosis of obstruction, for it is natural that respiratory pressure changes should be transmitted through the wall of a thin-walled cavity. Lipiodol (a few cubic centimeters are sufficient), should be injected into the cavity and watched under the screen. Immediate films should be taken before the patient has a chance to cough out the lipiodol if the cavity is open. If it is blocked, the lipiodol will outline its dimensions perfectly when the patient is moved about in various positions. The lipiodol may be followed by injection of a few cubic centimeters of gentian violet or methylene blue, which the patient should be told to watch for in his sputum.

The patient should be made to strain and to sniff or suck against the closed glottis. With a stop-valve opening cavity pressure should remain high or continue to rise as long as the patient strains; sniffing or suction should produce but a short drop in pressure, if any, and the drop should not continue to decrease while the patient continues to suck.

Opening the cavity to the outer world by means of a trocar or catheter may open a previously occluded bronchus or may release a stop-valve mechanism. Thus, some noncommunicating cavities developed a bronchial communication after they were drained (Cases 2 and 5).

CASE 2.—Mrs. C. P., aged twenty-five years, was first seen on Sept. 25, 1931. In February, 1929, she had expectorated one and one-half cupfuls of blood. A film was finally diagnosed by Garland in June, 1931, as evidencing bilateral tuberculosis with a small cavity at the right apex. The patient curtailed her work and took an inadequate cure until January, 1936, when she was put to bed. Expectoration was scanty, but the sputum contained tubercle bacilli. In films taken Feb. 24, 1936, a large cavity was apparent in the right infraclavicular region with a small amount of fluid at its base. Attempts at pneumothorax were unsuccessful and on Oct. 5, 1936, Rogers avulsed the right phrenic nerve. After phrenicectomy the cavity seemed to have disappeared. The patient had no cough and little expectoration. However, films taken on Dec. 16, 1936, again showed an annular shadow. The cavity measured 4 by 5 cm. and had a fluid level. On Dec. 28, 1936, a needle was inserted into it through the right sixth intercostal space posteriorly. Thick pus and air were withdrawn and 4 c.c. of lipiodol and a little methylene blue were injected. The lipiodol outlined the bottom of the cavity; none of it was seen to enter the bronchi. Films

taken the next day disclosed no communication with the bronchus. By turning the patient in various positions, the lipiodol which still remained in the cavity could be made to outline its borders; it measured about 6 cm. in diameter. Thinking that he might be dealing with a localized empyema pocket, Rogers inserted a second needle on Dec. 31, intending to substitute the purulent content with oil. However, on attempting to do this, the patient began to cough, so that the needle was withdrawn after a few drops of oil had been injected. A day later the cavity was again needled through a higher intercostal space. A small amount of pus was withdrawn followed by so much air that it was apparent that the previously blocked bronchial communication was now open. Methylene blue injected this time was promptly expectorated. On Jan. 6, 1937, the cavity was again needled, in order to determine whether there was an intermittent valve block. A few cubic centimeters of thick pus were withdrawn in which lipiodol and methylene blue, injected ten days before, could still be recognized. The pus was followed by a small amount of air, after which further attempts at aspiration yielded nothing but a little bloody fluid. It seemed as though the bronchial opening were temporarily closed. Films taken immediately afterward showed that the cavity contained about one-half as much lipiodol as it had on Dec. 29.

On Jan. 9, 1937, it was decided to attempt closed suction drainage in the hope of avoiding thoracoplasty. The notes read:

"This patient has a tuberculous cavity on the right side which apparently communicates only intermittently and partially with the bronchus. The cavity had been needled by Dr. Rogers and methylene blue and lipiodol had been injected into it. The patient never expectorated the lipiodol. On one occasion she expectorated methylene blue some twenty-four hours after it was introduced and on one occasion not at all. A needle introduced into the cavity and sucked on with a syringe yielded a small amount of air withdrawn with difficulty, i.e., under considerable suction. With this in mind, therefore, it was thought that could the noncommunicating cavity be collapsed by suction or would it collapse by opening it, the patient might avoid a thoracoplasty. This was all the more to be desired as her vital capacity was low (1,300 c.c.) and it was likely that if collapse were to be attained by a thoracoplasty the thoracoplasty would have to be an extensive one. It is decided to attempt to introduce a trocar into the cavity and to follow the trocar with a catheter.

Procedure.—This is done under the fluoroscope under novocaine. It proves unexpectedly difficult to hit the cavity accurately with a trocar and one is successful only after the second attempt. After the trocar is in place, pus and blood come from it. The bleeding amounts to perhaps an ounce or so in all. Introduction of the trocar or of the catheter opens the bronchial communication, for the patient immediately begins to expectorate blood and lipiodol which she had never done before. An x-ray taken immediately afterward shows that the lipiodol has been sprayed by cough into the upper part of the left lung."

The patient was not upset by the operation, but no reduction took place in the size of the cavity. If the cavity was totally blocked before introduction of the catheter, it was now blocked by a valve mechanism. On Jan. 19, 1937, lipiodol and air were injected into the catheter under the screen; when air was injected the cavity dilated; it did not contract when suction was applied. A valve mechanism was therefore apparent.

Suction drainage seemed futile and its continuance perhaps dangerous. On Jan. 25, 1937, thoracoplasty was decided upon. The notes read:

"This patient had a cavity in the middle of the right lung field. The cavity was closed by a valve which admitted air, but did not let it out. This was shown by the fact that when she coughed pressure was produced in this cavity, but when she sucked, scarcely a variation of the manometer took place and no negative pressure

was attained. An attempt had been made with this in mind to close the cavity by introducing a drain, thinking that were the air allowed to escape externally the cavity might collapse. However, no such collapse was apparent. It is therefore thought advisable to attempt closure of this cavity by thoracoplasty.

An incision is therefore made in the ordinary manner carefully avoiding contact with the site of the previous drainage opening from which the catheter had been temporarily withdrawn. With some difficulty the first three ribs are resected. The apex is then dropped after the manner of Semb. After thoracoplasty the lung dropped downward, so that the intercostal drainage tube traversed the chest wall, but no longer entered the lung."

The patient reacted to this operation with a rise in temperature to 38.5°. The wound healed without suppuration.

On Feb. 7, 1937, the cavity was still outlined behind the clavicle, being slightly smaller than before operation.

On February 13, 1937, the second stage of thoracoplasty was carried out. The notes are as follows:

"A vertical limb is added to the angle of the incision and the thus formed triangular flaps are retracted. The sinus leading to the lung has completely healed. Above the apex is a large, air-containing space without any exudate. It, fortunately, has remained uninfected. Eight and one-half cm., 8½ cm., and 6 cm. are removed of the fourth, fifth, and sixth ribs. Part of the transverse processes of the fourth and fifth vertebrae are also removed. The intercostal nerve-vessel bundles are much thickened and there are intercostal lymph nodes the size almost of a lima bean. The apex of the lung felt in the wound is much indurated. Under its dome posteriorly there lies a cavity which can be indented. On account of the great risk of causing a perforation at this point no further separation is attempted. A drain is introduced into the wound on account of the large air-containing, extrapleural space at the apex. The wound bed following resection is dusted with iodoform powder. The ribs are removed with a galvanocautery."

The patient made a smooth convalescence; cough and sputum decreased. Films taken on March 1, 1937, showed the right upper lobe to be more completely collapsed and the cavity to be smaller; droplets of lipiodol were still visible scattered through the collapsed portion of the lung. The patient was discharged to her home on March 9, 1937. The temperature has remained normal. The sputum has disappeared and no tubercle bacilli have been found since December, 1936. Films of April 15 no longer demonstrate a cavity; there are still a few droplets of lipiodol scattered through the right upper lobe.

CASE 3.—J. R., a man twenty-three years of age, was admitted to the San Francisco Hospital in April, 1935, and discharged to the Hassler Health Farm. He expectorated an occasional streak of blood; otherwise he had no sputum for weeks. Films gave evidence of a large cavity in the right lung. Pneumothorax was tried without success. He was readmitted to the San Francisco Hospital on Sept. 4, 1936. Tubercle bacilli were first noted in the sputum on Sept. 8, 1936. On Oct. 31, 1936, a cavity was needled through the second intercostal space; air was withdrawn. Pressure in the cavity on quiet respiration read -1½ + 1½; forced breathing produced wider oscillation. Two cubic centimeters of lipiodol and a little methylene blue were injected. The patient never coughed out either of these substances; lipiodol was still demonstrable in the cavity on Feb. 16, 1937, when a thoracoplasty was done of the upper three ribs. The cavity and its lipiodol content remained unchanged. On March 18, 1937, the fourth to seventh ribs and the fourth to sixth transverse processes were resected. Films of March 30 still demonstrated a cavity 3 cm. in diameter, with lipiodol still in it.

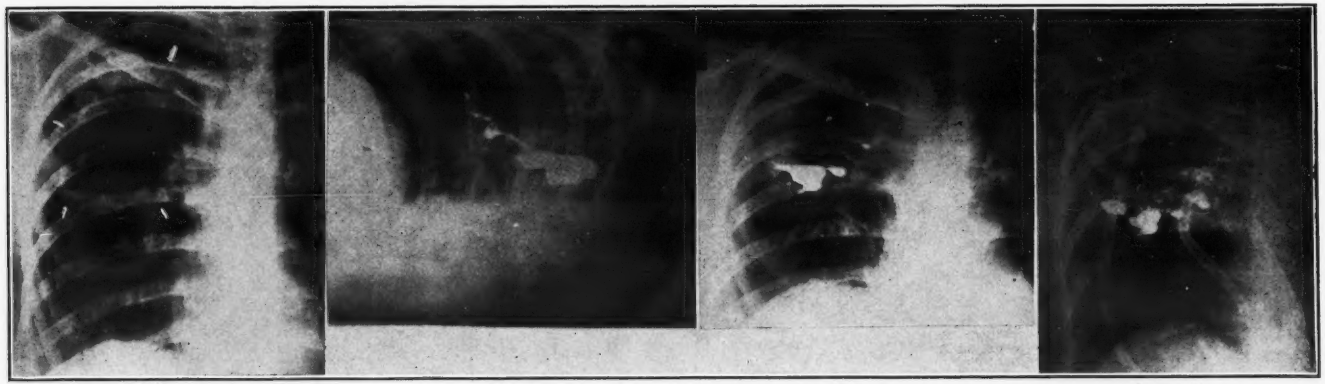


Fig. 7. Fig. 8. Fig. 9. Fig. 10.

Fig. 7.—Case 2. Feb. 24, 1936: large annular shadow with fluid level, upper right chest.

Fig. 8.—Case 2. Dec. 29, 1936: lipiodol injected into cavity through chest wall; patient lying on left side; fluid level seen above lipiodol; some lipiodol has entered the bronchial tree.

Fig. 9.—Case 2. Jan. 6, 1937: patient upright; lipiodol still remains in cavity eight days after injection; no lipiodol seen in bronchial tree.

Fig. 10.—Case 2. Jan. 10, 1937: cavity drained with rubber tube; lipiodol in cavity.

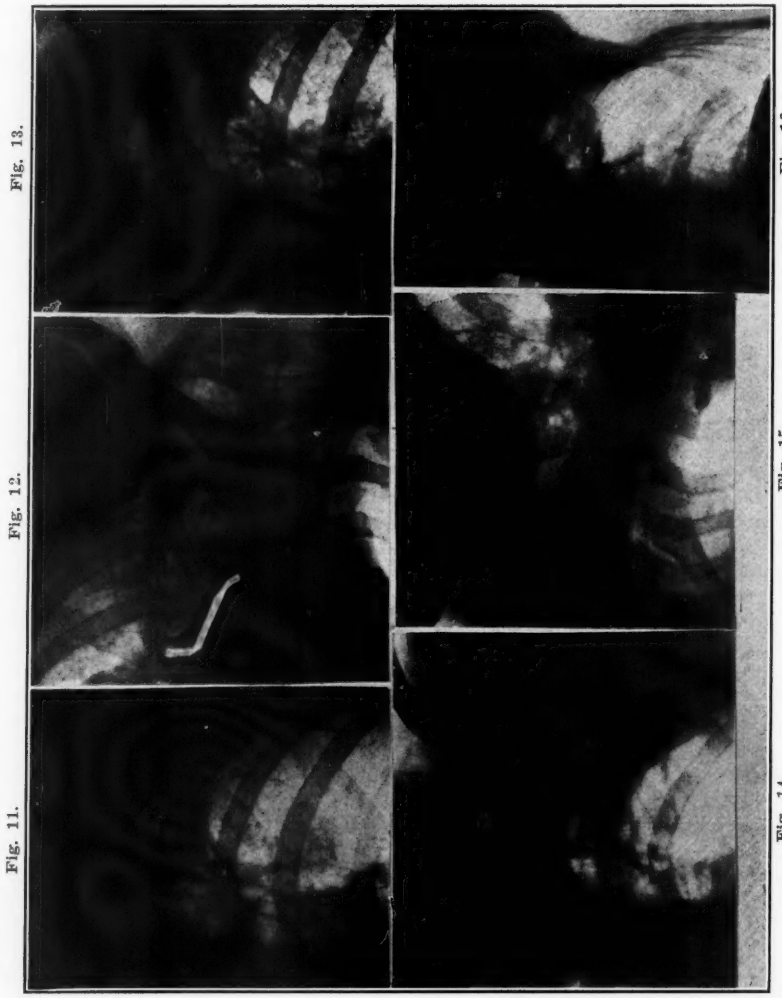


Fig. 11. Fig. 12. Fig. 13. Fig. 14. Fig. 15. Fig. 16.

Figs. 11-16.—See opposite page for legends.

BRONCHOSCOPY

Bronchoscopic attempts at determining whether a tuberculous cavity is open or closed are less likely to yield definite information than the x-ray or the aspirating needle. Apical bronchi are not visible in the bronchoscope; an entrance to cavities lying in the lower lobes may be visible, yet application of cocaine or adrenalin, suction or swabbing necessary to clear the bronchial field, will raise doubt whether the bronchi were open before introduction of the bronchoscope or not. It is probable that both needling and bronchoscopy may open a previously blocked bronchial communication. Release of pressure in an inflated cavity may open a blocked exit, as emptying a distended urinary bladder may relieve a valvelike closure at the bladder neck; bronchoscopic shrinkage and aspiration may relieve bronchial obstruction here as elsewhere.

Filling a cavity with lipiodol via the trachea is evidence that the bronchus leading to it is open in one direction at least; spilling of lipiodol out of the cavity denotes that it is open in the other. However, one will hesitate in tuberculous patients to use the quantity of lipiodol necessary for such a demonstration.

Salkin, Cadden, and McIndoe[9] state that physical signs of cavitation may persist even when a cavity is closed. I have made no studies in this regard.

TREATMENT

Most observers disagree with Coryllos' contention that closure of a draining bronchus leads to a cure of tuberculous cavities. The subject has been sufficiently discussed before this Association. It is true that a closed tuberculous focus offers a better prognosis than an open one, yet obstruction of a draining sinus from an open tuberculous abscess often leads to fever, toxicity, and disaster. Secondary infection plays an important, often a decisive, rôle. Similar considerations apply to the experiments of Adams and Singer,[1] who found that tuberculous infection ran a milder course in obstructed atelectatic lobes of dogs than it did in open ones.

Indeed, if one may judge from the few cavities studied with methods just described, it would seem that partial obstruction is not only a frequent cause of cavitation, but a frequent obstacle to cure, both by natural and artificial means; and that total permanent obstruction, if there is

---

Fig. 11.—Case 3. Oct. 31, 1936: lipiodol injected through chest wall into large cavity of the right apex; fluid level above lipiodol; patient upright.

Fig. 12.—Case 3. Oct. 31, 1936: same as shown in Fig. 11, with patient lying on left side.

Fig. 13.—Case 3. Jan. 18, 1937: cavity still contains lipiodol, two and one-half months after injection; patient upright.

Fig. 14.—Case 3. March 4, 1937: after thoracoplasty; cavity has no fluid level; it is possible to diagnose it only from the lipiodol still remaining in it four months after injection; patient upright.

Fig. 15.—Same as shown in Fig. 14, with patient lying on right side.

Fig. 16.—Case 3. May 12, 1937: lipiodol still seen seven months after injection at the bottom of a homogeneous apical density, representing a blocked cavity.

such a thing, is also a factor that causes cavities to resist attempts at closure. It is because their bronchial outlets are stenotic that some cavities persist; this thesis seems to explain the failure of some of our attempts at treatment sufficiently well to be offered for your consideration at this time. We have come to look upon our methods of operative treatment as means of allowing natural forces, which tend toward shrinkage and collapse, to act upon the walls of a cavity. We have looked upon cavities as being held open by more or less direct attachments to the bony thorax and have thought that if these attachments are severed or loosened, or if the chest wall is permitted to cave in, conditions would be created which would allow a cavity suddenly to collapse or more gradually to shrink. This idea underlies attempts at obliterating cavities by pneumothorax, by pneumolysis, by the various so-called collapsing operations, and especially by apicolysis and similar operations as advocated by Semb.[18] The conception seems reasonable for cavities with a free bronchial opening.

If, however, the opening is not free, so that each breath tends to inflate the cavity, may not operations that loosen it from its attachments tend rather to increase than to decrease its size, or tend, at best, to displace it in toto from its original position rather than to cause it to collapse? Especially should displacement follow operation in which a lobe containing a cavity is freed after the method of Semb or by intrapleural pneumolysis. If future investigation and observation should corroborate the suspicion that cavity block is more frequent than it appears to be and if such a block explains the failure of collapsing treatment to close certain cavities, then other methods of attack would seem necessary and an accurate diagnosis of the relation of a cavity to the bronchial tree be highly desirable in making a proper choice of procedure.

CASE 4.—Illustrating the effects of detaching a cavity from its surroundings is the case of a thirty-eight-year-old baseball player, R. K., who entered the San Francisco Hospital on May 27, 1936. He was found to have a cavity at the right apex, 6 cm. in diameter. His temperature averaged 101°; the sputum varied from 15 to 30 c.c. per day and contained tubercle bacilli. Pneumothorax was begun July 20, 1936, with an initial reading of -3 -1. The apex was adherent and the cavity remained unchanged, while the sputum increased from 20 to 40 to 60 c.c. per day, so that pneumothorax was discontinued on August 17, 1936. On Aug. 27, 1936, the first three ribs were resected and the lung was loosened after the manner of Semb. After loosening it one saw that the apex consisted of a thin-walled cavity that ballooned in and out with respiration (after apicolysis therefore the bronchus seemed to be open). The operation had no effect on the size of the cavity; it merely displaced it downward. On Sept. 17, 1936, lipiodol was instilled into the trachea. It could not be made to enter the cavity. A week later, Sept. 24, 1936, the cavity was needled; on quiet respiration pressure in it measured -4 +3. Forced expiration blew the water out of the gauge at a reading of more than +12. Lipiodol and dye were introduced through the needle. The dye first appeared in the sputum

nine hours later. Delay in expectoration of the dye and failure of the intratracheal lipiodol instillation to reach the cavity aroused suspicion that the cavity might be intermittently blocked, although it was certainly not permanently so. It was thought that an intermittent block might be the reason that thoracoplasty and apicolysis failed to reduce its size, but merely displaced it downward. With this in mind it was decided to try to introduce a catheter into it and to make suction drainage in the hope of collapsing it. Accordingly, on Oct. 10, 1936, a catheter was inserted through a trocar in the fifth intercostal space posteriorly, through which several ounces of pus, swarming with tubercle bacilli, were withdrawn. The catheter was connected to a Connell suction apparatus. The basal pneumothorax, which still persisted, was obliterated by removing 1,500 c.c. of air from it two days later.

Fig. 17. Fig. 18.

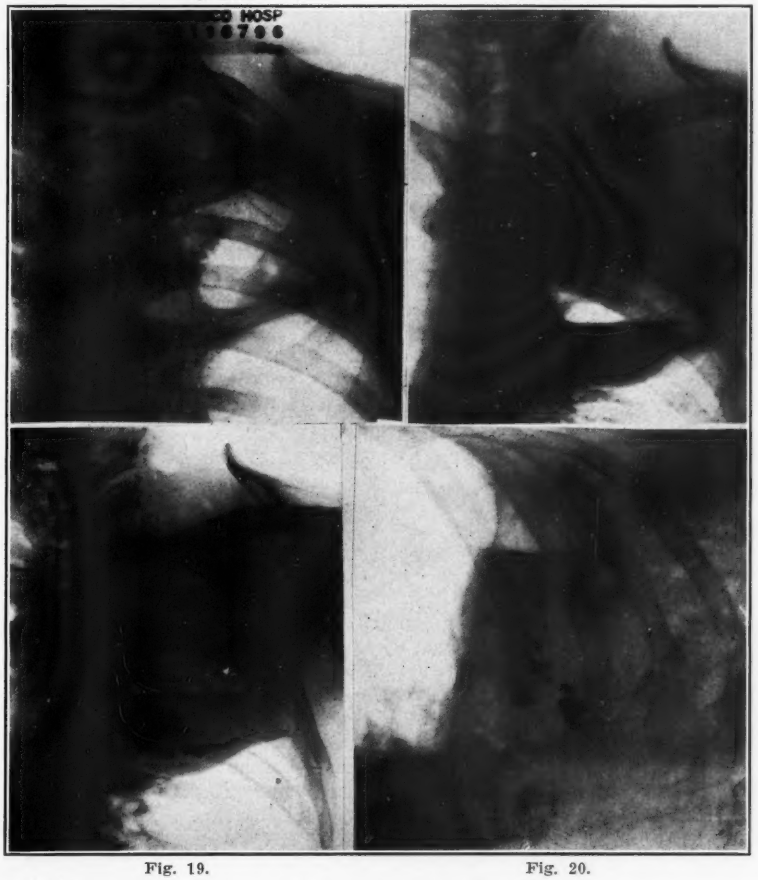


Fig. 19. Fig. 20.

Fig. 17.—Case 4. July 14, 1936: large cavity at right apex.

Fig. 18.—Case 4. Sept. 17, 1936: three weeks after apical thoracoplasty and apicolysis after Semb; cavity not diminished in size but displaced downward.

Fig. 19.—Case 4. April 24, 1936: cavity injected through chest wall with lipiodol patient upright.

Fig. 20.—Case 4. Sept. 24, 1936: same as shown in Fig. 19, with patient reclining on left side.

Neither this cavity, which was possibly though not certainly blocked, nor Mrs. C. P.'s cavity (Case 2), which was certainly so, was reduced in size by suction drainage. In both of them drainage had merely the effect of opening the bronchial communication and making further attempts at suction futile.

R. K. (Case 4), wore his tube five months, got a subscapular abscess, and retained an external bronchial fistula two months after removal of the tube. Mrs. C. P.'s sinus promptly closed after the tube was removed and subsequent thoracoplasty seems to have closed the cavity.

Suction drainage was tried without success in one other desperately ill boy.

CASE 5.—J. D., aged twenty-six years, came under Pierson's care in November, 1935. He had multiple tuberculous cavities throughout the right lung, had high fever, and was toxic. Pierson attempted artificial pneumothorax in December, 1935. It was unsuccessful; the air collected in pockets. The right upper lobe was nearly airless, but contained an uncollapsed cavity. The interlobar fissure lay high, so that Newell made a roentgenologic diagnosis of atelectasis of the right upper lobe through bronchial obstruction. A large cavity in the middle right lung field was needled under the screen on Dec. 10, 1936. Lipiodol and methylene blue injected into the cavity were not expectorated and did not leave the cavity when the patient was turned on his left and right side. Following the needling a trocar was introduced into the cavity under the screen and a catheter inserted. A syringe was attached to the catheter. The cavity was seen to get larger and smaller as the plunger of the syringe was moved in and out. The catheter was connected to a Sprengel's water pump, which, by means of a water trap, kept a constant suction drainage at a level of about -10 cm. The boy was not improved; indeed, the extent and toxicity of his process made it unlikely that he would be; it was thought, however, that suction might alleviate his constant cough and profuse expectoration.

On Dec. 24, 1936, his parents took him home, attempting to install a water pump apparatus under their own supervision. The apparatus was constantly clogged with pus. The boy died on Feb. 3, 1937. Autopsy was refused.

Two other blocked and tightly adherent cavities were treated by open drainage.

CASE 6.—J. B., a boy of fifteen years of age entered the San Mateo Community Hospital July 10, 1931, with a right upper lobe cavity and about two ounces of sputum per day, which contained tubercle bacilli. Pneumothorax was instituted; later the phrenic nerve was avulsed. The upper lobe was adherent, but the cavity apparently closed and the boy was discharged in September, 1933, with no sputum. In January, 1934, the cavity reopened and an infiltrate appeared in the left upper lobe. First a left, then bilateral, pneumothorax was begun. The right apical cavity, which lay in an airless and bronchostenotic right upper lobe, was uninfluenced by phrenectomy. A first-stage thoracoplasty was done on Nov. 26, 1934, a second stage on April 20, 1935. The patient still retained about 20 c.c. of sputum containing tubercle bacilli. On Oct. 5, 1936, the cavity was opened and packed with iodoform gauze. Air came from the cavity at the time of operation and at this time the communicating bronchus was open in one direction at least. The patient recovered and gained considerable weight. A bronchial fistula formed, which remains open. The patient still retains 8 c.c. of sputum in which tubercle bacilli are to be found.

CASE 7.—A. L. entered the San Francisco Hospital with bilateral apical cavities on Feb. 10, 1931. The right apex was compressed with a wax filling on Aug. 27, 1931. The pleura was thin and the lung movable. It ballooned in and out on respiration. On Sept. 26, 1931, the left apex was compressed with wax. The left lung was also soft and movable. The patient's sputum was scarcely diminished by these operations. Suspecting an uncompressed cavity above the wax at the right top, a needle was inserted under the fluoroscope on Dec. 17, 1931. One to 2 c.c. of thick pus were withdrawn and lipiodol injected. By August, 1932, the sputum had decreased to about 10 c.c. per day. The lipiodol injected eight months before was still to be seen. The patient was transferred to Weimar Sanatorium, where he began to expectorate the wax. On Oct. 31, 1935, the wax was removed through an axillary incision. A small bronchial fistula was seen. The wound was packed with iodoform gauze. The next day he had a massive subcutaneous emphysema and a temperature of 102°. The bronchial fistula persisted. A small tortuous sinus leading to a cavity containing 20 c.c. of pus was excised Oct. 14, 1936.

On Jan. 14, 1937, lipiodol injected into the sinus failed to demonstrate a bronchial connection; the sinus closed and remained so. Cough and sputum still persist.

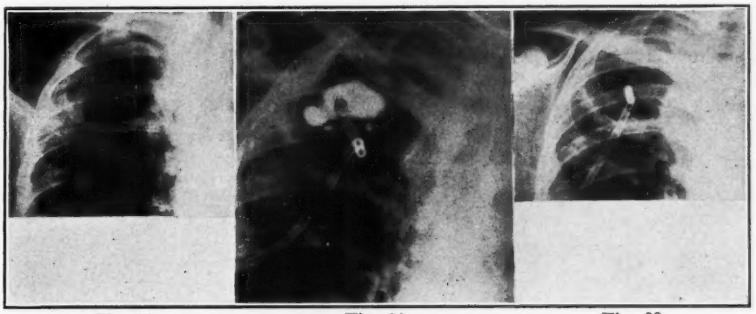


Fig. 21. Fig. 22. Fig. 23.

Fig. 21.—Case 5. Large right side apical cavity with annular shadow.

Fig. 22.—Case 5. Dec. 10, 1936: cavity drained by rubber catheter; lipiodol injected, patient recumbent.

Fig. 23.—Case 5. Dec. 19, 1936: cavity drained by catheter; lipiodol no longer remains.

Four patients with accurately diagnosed blocked cavities were treated by ordinary thoracoplasty—how many of my older patients with thoracoplasty may have had blocked cavities I do not know. Mrs. C. P., with a blocked cavity treated by thoracoplasty, is described above as Case 2.

CASE 8.—Miss L. McL., aged twenty-seven years, had a cough and expectoration of tubercle bacilli dating from March, 1935. She entered the San Francisco Hospital on June 17, 1935. She had a large right apical cavity which pneumothorax failed to collapse. Anterior and posterior thoracoplasty were done on March 17 and April 9, 1936. The cavity was thought to have been closed. The sputum decreased to 2 to 5 c.c. per day; tubercle bacilli could no longer be demonstrated and the patient was discharged on June 26, 1936. She felt well until Jan. 1, 1937, when after an attack of influenza the sputum increased in amount to 10 c.c. per day and contained tubercle bacilli. She reentered the San Francisco Hospital on Feb. 11, 1937, where a cavity of the right apex was demonstrated at the site of the old thoracoplasty.

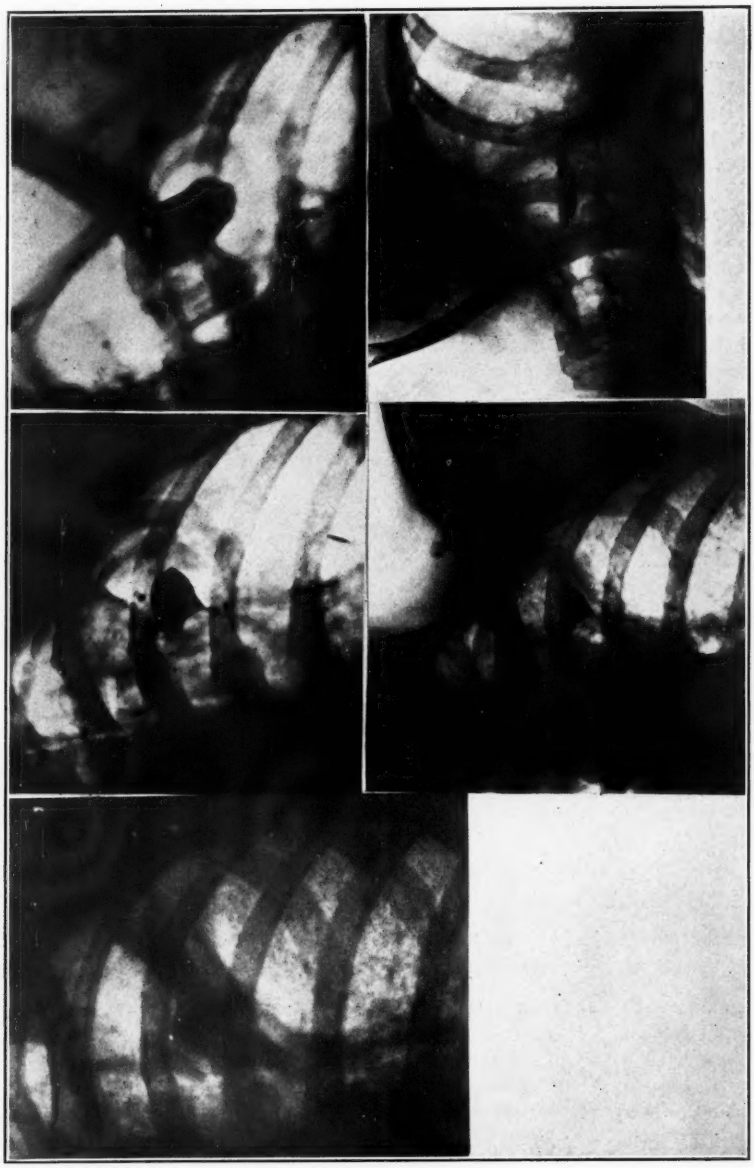


Fig. 24. Fig. 25. Fig. 26. Fig. 27. Fig. 28.

Figs. 24-28.—See opposite page for legends.

On Feb. 18, 1937, the cavity was needled. The manometer showed no oscillation either on quiet breathing or with cough. Five cubic centimeters of lipiodol and 1 c.c. of methylene blue were injected which outlined the cavity but could not be made to run into a bronchus. Dye appeared in the sputum thirty-six hours later. Revision of the former thoracoplasty with extensive removal of newly formed bone and resection of two further ribs (fourth and fifth) was done on March 13, 1937. Films taken on April 23, 1937, no longer show a cavity. The sputum averages 0 to 3 c.c. per day and tubercle bacilli are no longer demonstrable in it.

The third case of thoracoplasty for blocked cavity is described as Case 3.

CASE 9.—B. E. had a nonproductive cough since 1931. In December, 1933, she had a small hemorrhage. X-rays disclosed a tuberculous cavity at the left apex. Pneumothorax was begun in February, 1934, and continued for six months. The cavity remained unchanged. On Sept. 29, 1933, the left phrenic nerve was avulsed. The cavity still remained. On July 3, 1935, the first three ribs were resected and on July 17, 1935, the fourth and fifth. Some three weeks later a subscapular abscess formed, which was drained; the pneumothorax cavity, which still persisted at the base of the left chest, fortunately was not infected. Cough, which had been absent, returned and x-rays taken at this time showed that the cavity at the left apex had either recurred or had never been closed, for it was now larger than ever.

Lipiodol was injected under novocaine infiltration. The patient began to cough and tasted the novocaine; the lipiodol, however, remained and was not expectorated. On Oct. 29, 1935, films were taken in various positions outlining the cavity; twenty-four hours later the patient was again fluoroscoped. The lipiodol still remained in the cavity. Although the patient stated that she could note the odor of lipiodol, none had left the cavity, nor could it be made to spill out however one turned the patient. Part of it still remained on Dec. 12, 1935, some six weeks after injection and in spite of an extensive renewed apical thoracoplasty done on Nov. 1, 1935. She returned to the California Sanatorium at Belmont after the third extensive apical thoracoplasty. The cavity persisted and the patient continued to have a cough and sputum. In April, 1937, she had a hemorrhage of about six ounces.

On May 8, 1937, the cavity was again needled under the fluoroscope. The needle was inserted through the back; with the patient prone, the manometer read +1, +1½ on quiet respiration; when the patient sniffed, the readings were -3; on coughing, +4. Methylene blue and lipiodol were injected. The patient expectorated neither the dye nor the lipiodol, and from films taken May 11, 1937, it was reported that no lipiodol had left the cavity.

On May 17, 1937, part of the wound overlying the cavity was reopened and regenerated bone resected. The cavity was encountered lying close to the posterior chest wall. It measured about 3 × 2 × 2 cm., was thin walled, and had a few fine bronchial openings running into it. Its posterior wall was freed and iodoform gauze packed down onto this loosened wall in the hope that the cavity might remain compressed. The patient reacted to the operation with a temperature of 38°. August, 1937: The last operation abolished cough and expectoration. The sputum is now negative for the first time in years. The bronchial fistula has closed.

Fig. 24.—Case 8. Nov. 18, 1935: large right sided apical cavity.

Fig. 25.—Case 8. Feb. 18, 1937: ten months after thoracoplasty; cavity remains open; lipiodol injected into cavity through chest wall.

Fig. 26.—Case 8. Feb. 18, 1937: same as shown in Fig. 25.

Fig. 27.—Case 8. Feb. 18, 1937: lipiodol still remains in cavity; patient upright.

Fig. 28.—Case 8. Same as shown in Fig. 27, with patient lying on right side.

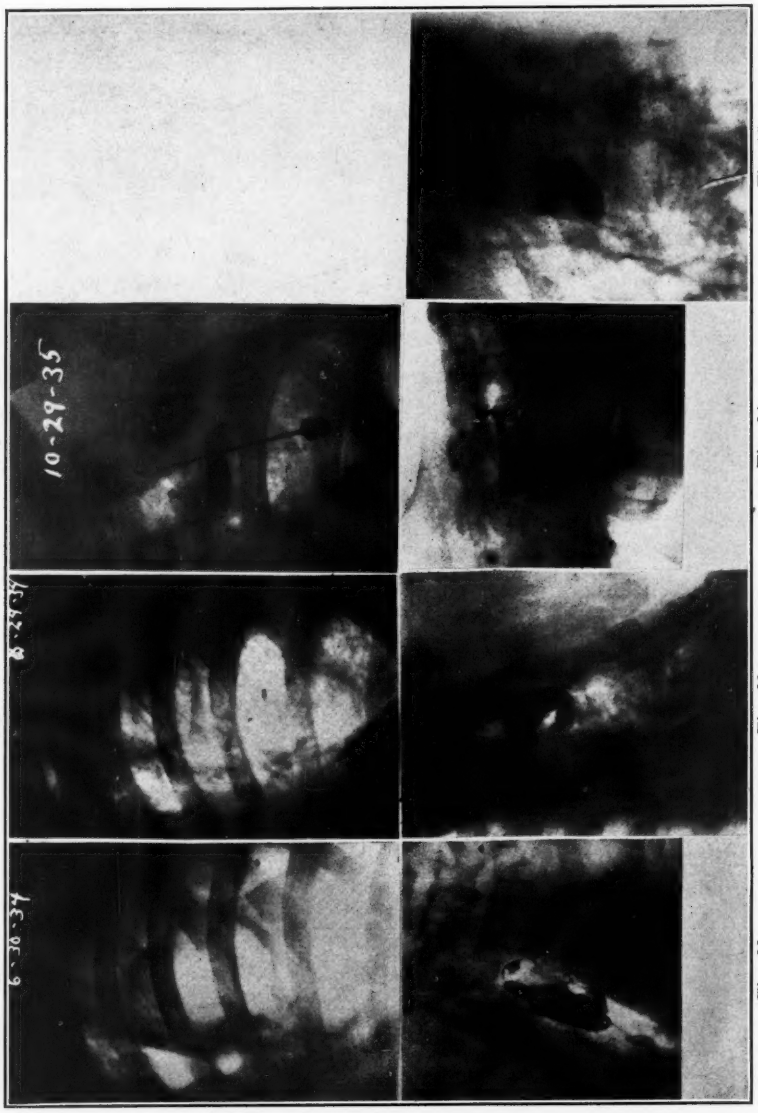


Fig. 29. Fig. 30. Fig. 31.

Fig. 32. Fig. 33. Fig. 34. Fig. 35.

Figs. 29-35.—See opposite page for legends.

This series is small. Formal attempts at accurate diagnosis in cavities suspicious of being obstructed are of recent date. The diagnostic efforts which have been presented may be of more interest than the efforts at treatment, which have brought nothing new. Closed suction drainage of blocked cavities, a method which at the outset seemed most promising and reasonable, has been a failure.

Six of these 9 patients were treated by fairly extensive thoracoplasty. Two seem to have been cured; 4 retain their cavities. The operations are too recent to judge of final results. In one cure (Case 8) extensive revision was necessary; another (Case 2) was done after suction drainage had failed to close the cavity. The effect of thoracoplasty is hard to explain; perhaps it is truly a compressing rather than a collapsing operation when done for inflated cavities; perhaps compression prevents their further inflation or perhaps it opens an occluded bronchus, permitting the cavity to empty. It is difficult to conceive of pressure upon a tuberculous focus doing it anything but harm.

Addition of apicolysis to the usual thoracoplasty seems inadvisable. Apicolysis seems to accomplish nothing but displacement of the blocked cavity downwards. It is reasonable to suppose that such a cavity would be more easily closed by thoracoplasty when the lung is adherent than when it is loose.

Open drainage with iodoform gauze packing has succeeded in curing the tuberculous process in two tightly adherent, isolated apical cavities, as far as the cavities are concerned (Cases 6 and 7). However, both patients still retain tuberculous sputum because of other pulmonary foci. A third obstinate cavity, treated by open drainage, was opened but two months ago. This patient is now free of cough and expectoration. What little sputum there is contains no tubercle bacilli; a fourth, successfully treated in this manner, is not counted here because of insufficient proof of obstruction.

Although open packing has converted two obstinate tuberculous cavities into perfectly clean, epithelialized areas, the patients retain bronchial fistulas and cumbersome dressings. The fistulas will be difficult to close; as usual in the open treatment of pulmonary tuberculosis,

---

Fig. 29.—Case 9. June 30, 1934: large left apical cavity, uncollapsed, in spite of pneumothorax; apical adhesions.

Fig. 30.—Case 9. Aug. 29, 1934: pneumothorax absorbed; cavity remains, seen as large, thin-walled annular shadow. It has changed its size and position since June, 1934.

Fig. 31.—Case 9. Oct. 29, 1935: three months after thoracoplasty; cavity persists; it has been needled and injected with lipiodol, none of which enters the bronchial tree.

Fig. 32.—Case 9. May 8, 1937: eighteen months after third extensive thoracoplasty; large cavity persists in apical scar; lipiodol injected; patient recumbent.

Fig. 33.—Case 9. Same as shown in Fig. 32, with patient upright.

Fig. 34.—Case 9. Same as shown in Figs. 32, 33, with patient lying on right side.

Fig. 35.—Case 9. Same as shown in Figs. 32-34, with patient in Trendelenburg position. In none of these last four films does lipiodol leave the cavity.

the lung has retreated as the cavities were packed and healed and what was originally a little hole has become a very big one.

Should further experience show that this small series is not representative, should suction drainage of blocked cavities prove a failure (and this seems probable), should larger series of thoracoplasty prove that the apparently favorable course of these two cases is not consistent, should open drainage and packing (applicable to but a few strictly isolated cavities) prove too risky and too objectionable, there still remains another mode of attack—excision of the blocked tuberculous cavity by partial or total lobectomy. The operation is tempting, especially for those apical cavities which so impudently protrude into a pneumothorax out of an otherwise collapsed lung. Excision, if successful, would be the ideal method. The day may be at hand when it will be safe; hitherto it has seemed too risky to justify trying it.

SUMMARY

1. Tuberculous bronchial stenosis and especially stop-valve stenosis play a rôle in the production of excavation.

2. Destructive ulceration of the more rigid pulmonary framework and inflation of the resilient lung lead to excavation.

3. Cavities produced in this way are spherical ("annular shadows").

4. Diagnosis of obstruction of a cavity is discussed: (a) by means of radiography with and without opaque substances, (b) by needling, and (c) by bronchoscopy.

5. Manometric pressure changes in blocked cavities are discussed.

6. Illustrative cases are cited and methods of treatment are discussed: (a) suction drainage, disappointing; (b) thoracoplasty, doubtful; (c) thoracoplasty with apicolysis, inadvisable; (d) open packing, curative but restricted in application and troublesome; (e) possibility of resection of the cavity or lobectomy is considered.

REFERENCES

1. Adams and Singer: Am. Rev. Tuberc. **31:** 373, 1935.
2. Coryllos: Am. Rev. Tuberc. **33:** 639, 1936.
3. Graef: Ergebn. d. ges. Tuberk.-Forsch. **7:** 257, 1935. (An excellent review with most complete bibliography.)
4. Moolten: Arch. Path. **19:** 525, 1935.
5. Reinders: Ztschr. f. Tuberk. **51:** 438, 1928.
6. Heinbecker: J. Clin. Investigation **4:** 459, 1927.
7. Jackson, C., and Jackson, C. L.: Pennsylvania M. J. **7:** 740, 1934.
8. Miller, Robt. T., Jr.: Arch. Surg. **12:** 392, 1926.
9. Salkin, Cadden, and McIndoe: Am. Rev. Tuberc. **34:** 364, 1936.
10. Hall: Lancet **1:** 61, 1922.
11. Blyth-Brooke: Lancet **2:** 240, 1931.
12. Pearson: Brit. M. J. **1:** 380, 1930.
13. Morland: Lancet **2:** 1311, 1933.
14. Sanes and Kenny: J. Pediat. **5:** 51, 1934.
15. Korol: Am. Rev. Tuberc. **31:** 475, 1935.
16. Tchertkoff and Bobrowitz: Quart. Bull. Sea View Hosp. **1:** 173, 1936.
17. Fischel: Am. Rev. Tuberc. **28:** 411, 1933.
18. Semb: Acta chir. Scandinav. **78:** 1, 1935.

THE FREE TRANSPLANTATION OF FAT FOR THE CLOSURE OF BRONCHOPULMONARY CAVITIES (LATTICE LUNG)

HAROLD NEUHOF, M.D.
NEW YORK, N. Y.

THE transplantation of fat into pulmonary cavities in order to promote their closure is an apparently new procedure. The concept is also illogical. For many years I have been interested in some experimental aspects and have practiced some of the clinical methods, of tissue transplantation. In a monograph on the subject of the transplantation of tissues, published in 1923, I indicated the criteria which I, like others, believed necessary in order to secure successful free tissue grafting. In essence they were a sterile field, a healthy receptive bed, and one capable of some degree of immobilization after transplantation. The fact that the operation which I have practiced and wish to advocate has succeeded in a field which meets none of these presumably essential requirements, proves its empirical basis. That the exceedingly simple procedure has aided in the solution of a problem which otherwise may be difficult or even impossible of solution, is the reason for its presentation at this time and place. The features of the lesions for which fat transplantation has been employed will be described briefly, the technique will be indicated, and the reports of seventeen cases in which the operation was employed will follow.

As the result of one of a variety of destructive lesions, there may be left in a smaller or larger portion of the lung, a defect with the following characteristics: Usually there is a communication with the skin, via an operative wound. The walls of the cavity are more or less mobile, the lining membrane is of reddish color, and there are trabeculae which cross hatch the interior. Vessels are to be noted in the trabeculated ridges. Between them are the orifices, often many in number, of bronchi of varying size. This is the picture generally termed lattice lung. The size of the lesion will vary, of course, in accordance with the extent of the destructive process. An important point is the fact that a lattice lung can be fully formed within a few weeks of the time of onset of the destructive bronchopulmonary lesion. Indeed, the appearance of the lining membrane of a lattice lung of a very few weeks' duration may correspond in every way to that of one of many years' duration.

The question of the nature of the destructive lesion which preceded the advent of the lattice lung, appears to be a matter of little moment. The lesion seems identical whether the cause was a preexisting putrid abscess of the lung or a nonputrid abscess with destructive features.

Read before the Twentieth Annual Meeting of the American Association for Thoracic Surgery, at Saranac Lake, N. Y., May 31, June 1, 2, 1937.

In addition to pathologic processes which lead directly to the formation of lattice lung, there are a number of forms of bronchopulmonary cavitation which are derived from other sources. Thus reference to the case reports discloses cavitation left after partial lobectomy, after the drainage of a tuberculous abscess of the lung, and after the removal of an infected echinococcus cyst of the lung. In no instance, however, was the lattice lung or analogous pulmonary lesion confined to the substance of the lung. In all cases the visceral pleura overlying the lesion either had been traversed deliberately for surgical purposes or had been perforated by spontaneous rupture of the intrapulmonary focus.

Spontaneous healing occurs in some cases of lattice lung. It is apt to take place when lesions are small, bronchial fistulas few in number, and when some coaptation of the walls exists. The tendency towards healing usually is to be noted at an early stage. However, even large postoperative cavities may heal most unexpectedly, apparently by some form of ingrowth of granulation tissue.

The problem of the closure or cure of lattice lung, or of open bronchopulmonary cavity, is one which may be difficult of solution. The solution does not appear to depend greatly upon the size of the cavity, although, of course, a huge cavity presents a more difficult problem than a small one. The closure of some small cavities, particularly those bearing but few bronchial openings, has upon occasion been accomplished by simple plastic flap measures. As more and more complicated cavities are encountered, with bronchial fistulas situated at inaccessible places between trabeculae, the cure becomes more difficult. Finally, when the lesion occupies a considerable portion of a pulmonary lobe, nothing short of a lobectomy has been apt to solve the problem. In my own experience, at any rate, lobectomy under those circumstances is not only a difficult but also a hazardous procedure.

As already stated, the idea of transplanting fat for the purpose of closure of bronchopulmonary cavities is ill conceived. The criteria for a successful free tissue graft are a sterile field, a bed capable of vascularization, and a field free from dead spaces, and one in which immobilization is feasible after transplantation. None of these criteria existed in any of the cases in my series.

It may be well to describe the first instance in which a fat graft was employed. This occurred in 1934. A child eleven months old was operated upon for an acute ruptured abscess of an upper lobe. There was a large communication between the empyema and the interior of the lung. The pulmonary cavity was smooth lined and presented trabeculae bearing pulsating vessels. There were numerous large and small bronchial openings. The cavity in the lung was loosely packed. It was evident that much of the upper lobe had been destroyed by the infection and that a lattice lung already existed at this early stage. The impression which was gained was that there was but little likelihood of cure

of the lattice lung by any procedure other than lobectomy. The decision was reached to attempt to fill the cavity at an intermediate stage, namely, when the acute infection had cleared up, and before secondary infection occurred. Various tissues were considered for purposes of free transplantation, but fat was decided upon because of bulk, ease of fit into recesses, and tendency towards viability with minimal blood supply. Two weeks after the drainage operation the cavity appeared reasonably clean and the fat graft was performed. A mass of fat about 2 inches long, 1½ inches wide, and one and a half inches thick, was detached from the buttock and laid into the cavity in the lung. It fitted snugly and reached up to the level of the excised portion of the rib. The margins of the skin were freed and strapped. The postoperative course was uneventful. Roentgen films revealed the existence of a homogeneous shadow in the operative field, the nature of which of course could not be determined. The shadow gradually disappeared in later films. The child has remained symptom free, and there has been no evidence of any tendency toward recurrence of the bronchopulmonary cavity. The view which I together with others hold concerning the ultimate fate of a free tissue transplant is that the graft disappears and is replaced by a more or less indifferent fibrous tissue. It is safe to assume that that was the fate of the fat graft, and it would be most unreasonable to believe that the graft remained viable. In any event, the result was a permanent healing of a lesion which in this case apparently would have been most difficult of solution by other methods.

Since the first operation was performed, fat has been transplanted for bronchopulmonary cavitation or for classical lattice lung in a variety of conditions and at different stages of the evolution of these lesions. The procedure also has been employed for purposes of hemostasis in otherwise uncontrollable hemorrhage from an infected pulmonary cavity. The details are to be noted in the case reports. An important consideration is the fact that if failure occurs after transplantation of fat, no appreciable harm is done.

TECHNIQUE

There is no preparation of the bed, namely, the floor of the cavity into which the graft should be placed. I no longer employ a single fat mass, but divide it into appropriate fragments in order to fit approximately the various recesses which often exist. In general, the larger the graft the less the likelihood of "take." Accordingly, the graft should be as small as will be compatible with the filling of the cavity. The transplant should not project above the rib level. In some instances it may be necessary to employ strips of gauze in the form of packs to hold the grafts in place. The treatment of the wound of the soft parts and skin depends upon the topographic features. In most instances I have strapped the skin edges together after a few sutures approximating the

soft parts. The skin should be thoroughly mobilized so that its margins can be brought together without any tension. In most instances, even in children, the operation of fat graft, obviously a very simple one, has been performed under local anesthesia. There were two cases (Cases 15 and 17) in which infection occurred, apparently following necrosis of some of the grafts, and yet healing followed. It is, therefore, important to realize that a bland infection following transplantation of fat should be treated by evacuation of the accumulated mucopus without the wide opening of the wound. The latter can always be done at a later stage if there is indubitable evidence of failure. It should also be noted that in several instances a bronchial blow existed for a short time after operation, to be followed by complete closure.

Concerning the results, complete healing occurred in eleven cases. The result could be termed a partial success or partial failure in three cases, and a complete failure in three. The sizes of the cavities varied greatly.

A perusal of the case reports will disclose the fact that lattice lung or bronchopulmonary cavities of varied origin were dealt with. In this small series one can find no evidence that the result was related to the nature of the underlying cause. Thus, there were two instances of transplantation of fat for cavities left after drainage of tuberculous pulmonary abscesses. The transplantation succeeded in one and failed in the other. Failure occurred after the transplantation of a large segment of fat for a lattice lung of longstanding (Case 7), but succeeded in another instance of equally longstanding (Case 4). There are two instances in which the transplantation cannot be properly characterized as one for either bronchopulmonary cavity or lattice lung. In Case 3 the fat graft was used in an open infected cavity for purposes of controlling hemorrhage. In Case 11 fat grafts were used for cavities opening on a flat surface. In neither of these instances could closure of the soft parts and skin be carried out. They are, however, included because no case in which a fat graft was employed has been excluded. Finally, there are several instances (Cases 5, 8, 16, 17) in which there was a fair possibility that spontaneous healing might ultimately have occurred. In these the successful grafting procedures led to a great shortening in convalescence. In all instances in which the fat grafts were successful, the resulting healed wound was a flat one. This is to be compared with the depressed scar seen usually after the healing of an opened pulmonary cavity.

CASE REPORTS (IN CHRONOLOGICAL ORDER)

CASE 1.—I. H., No. 364367, male, eleven months old, Mount Sinai Hospital, was operated upon April 13, 1934, for a pyopneumothorax of two weeks' duration. The roentgen film revealed several cascading fluid levels in an extensively involved upper lobe. At operation the encapsulated empyema cavity, opened by the excision of portions of two ribs beneath the scapula, revealed a large communication with the interior of the lung. The pulmonary cavity was extensive, smooth lined, and

presented many trabeculae bearing pulsating vessels. There were numerous larger and smaller bronchial openings between the trabeculae. The large central cavity had two main recesses, one extending beneath the scapula and the other anteriorly high up in the axilla to the level of the second rib. The cavity was loosely packed.

The comment made at the time of the operation was that much of the lobe had been destroyed, that a lattice lung already existed, and that an outlook for cure existed only if a lobectomy were to be performed. It was also noted that in this, as in other instances, there was the likelihood of secondary infection of the opened cavity. It was therefore decided to perform an operation intended to obliterate the cavity as soon as the acute infection cleared up.

The wound appeared clean within two weeks of the drainage of the pyopneumothorax, and a fat graft was performed on April 27. At the time of this operation the cavity was noted to be clean and had diminished somewhat in size, but presented the same large number of bronchial openings as noted at the original operation. Fortunately, for the purposes of the operation the child was plump and a fat mass of appropriate size could be obtained from the buttock. It was trimmed in keeping with the size and shape of the cavity and comprised a mass about 2 inches long and 1½ inches in the other diameters. The graft was laid in place in the cavity and fitted snugly. The skin margins were freed and approximated by adhesive straps. The postoperative course was uneventful. For practical purposes it can be stated that the graft healed in place, although one can assume confidently that progressive absorption took place. The wound has remained healed. The child has been symptom free, and the roentgen films are those of a normal pulmonary field. The last follow-up is of recent date.

Case 2.—A. S., No. 365580, female, two years old, Mount Sinai Hospital, was operated upon for acute putrid abscess of the lung following tonsillectomy. At operation on May 4, 1934, the lesion occupied much of the right lower lobe with several cavities and fluid levels. It was essentially bilocular with an intervening pulmonary bridge which was severed. There were numerous larger and smaller bronchial fistulas communicating with the bilocular cavities.

The convalescence was rapid and the wound soon assumed a healthy appearance. The cavity remained large without any great reduction in size. In view of the extent of the lesion, the likelihood of spontaneous healing appeared rather distant. A fat graft was decided upon because of the experiences in the first case. A mass of fat 2½ inches long and 1½ inches in the other diameters was removed from the buttock, and was trimmed and fitted into the pulmonary cavity. Because of a tendency toward expulsion of the graft, small gauze packs were placed over the graft, their ends being brought out at the angles of the wound. The skin was mobilized and approximated by strapping without tension.

The drains were removed after a few days. Primary union occurred. As in the previous case, the early postoperative films were those of "pneumonic infiltration." Whether the appearance in the film indicated a reaction to the grafts or the presence of the grafts is not known. Several months after operation the films were entirely negative. The child has been followed up to the present time and remains symptom free with healed wound.

Case 3.—W. H., No. 378071, male, forty-two years old, Mount Sinai Hospital, was operated upon April 9, 1935, for a chronic streptococcic abscess in the right upper lobe. The purulent discharge from the cavity was diminishing, but active recurrent hemorrhage from the wound began on April 18. When this could no longer be controlled by packing, a fat graft 3 inches by 2 inches by 2 inches was placed in the cavity and sutured in place. The sole purpose in transplanting the fat in this case was to control hemorrhage. Over the fat graft gauze packs were placed. Bleeding was controlled for a whole month by the procedure. Progressive

necrosis of the graft could be observed, and it finally became completely necrotic. Hemorrhage then recurred and a lobectomy was performed for hemorrhage on May 24. At the time of lobectomy the etiology for the chronic streptococcic abscess of the lung was found in a bronchostenosing carcinoma of the upper lobe bronchus.

CASE 4.—R. T., No. 379339, male, thirty-four years old, Mount Sinai Hospital. In 1930 a chronic putrid abscess in the left upper lobe was drained through a paravertebral incision. At no time was there a recurrence of the symptoms or signs of a pulmonary infection. However, occasional hemoptyses occurred. Two weeks before hospital admission, that is, in November, 1934, a brisk hemoptysis occurred and since that time smaller hemoptyses took place.

The roentgenographic and other forms of examination revealed a rather large multilocular cavity in the left upper lobe. At operation in November, 1934, the cavity was opened through the old scar, old blood clot was evacuated, some purulent fluid was noted, and the orifices of many bronchial fistulas were seen. The cavity was drained for three months, and was then permitted to close. Hemoptyses recurred. The patient was readmitted for the control of hemorrhage.

It was decided to reopen the cavity and to attempt its occlusion by a fat graft. At operation on May 3, 1935, the many-chambered, smooth-lined cavity with recesses in all direction was opened. Again numerous bronchial fistulas were noted. A fat mass, removed from the buttock, was 3½ inches long and 2 inches in the other diameters. It was divided into five appropriate sections in order to fit approximately the various recesses. The musculature, aponeurosis, and skin were closed in layers.

Expectoration of old blood in small quantities occurred for several days after operation. The wound healed by primary union. The patient was symptom free and remained well until his last follow-up on Feb. 18, 1936. He was apparently well physically when he committed suicide on June 8, 1936.

CASE 5.—G. F., No. 385080, male, thirty-seven years old, Mount Sinai Hospital, was operated upon Oct. 8, 1935, for an unusually large acute putrid abscess of the lung. The postoperative course was uneventful, but a large cavity remained. Indeed, the walls appeared to stiffen as time went on and the cavity assumed a distinctly chronic character. The lesion was a typical lattice lung with numerous bronchial orifices. Although it was realized that spontaneous healing might occur, a confirmed chronic lattice lung appeared to be the probable outlook. Accordingly, a transplantation of fat was performed forty-nine days after the drainage of the pulmonary abscess (Nov. 26, 1935). The fat mass removed from the buttock was 4 inches by 3 inches by 3 inches. It was divided into three sections which fitted the cavity snugly. The grafts reached to within half an inch of the skin level. The skin was freed and approximated without tension.

The postoperative course was asymptomatic. On the fourth day there was fever which rose to 101.6° but subsided the next day. The wound healed by primary union and has remained flush with the skin surface.

There have been no symptoms referable to the pulmonary lesion. On April 6, 1936, a bronchoscopy was performed and was negative. On May 27, 1936, a bronchogram was done and showed but one very small sacculation in one of the branches going to the former site of the abscess. The patient has remained well and was seen at a recent follow-up.

CASE 6.—R. M., No. 386733, male, nineteen months old, Mount Sinai Hospital, had a closed drainage of a tension pyopneumothorax performed on Nov. 13, 1936. Although the infection subsided, the escape of large quantities of air continued. The lung gradually became more and more collapsed with displacement of the heart to the opposite side. The wound was permitted to heal when the infection had cleared

up. It was hoped that absorption of the air in the closed pneumothorax would lead to gradual expansion of the lung. The persistence of the total or almost total pneumothorax led to the belief that a bronchopulmonary excavation existed. An exploratory thoracotomy was therefore decided upon, with a tentative plan to transplant fat into the cavity if such a procedure appeared feasible for purposes of closure. At operation, on Dec. 20, 1935, a large bronchopulmonary cavity was found in the posterior portion of the left lower lobe. There were several bronchial fistulas. The perforated pulmonary lesion extended upward to the interlobar fissure and was more extensive in the depths than on the surface.

There appeared to be three possible procedures for the purpose of cure. The first was lobectomy, the second was complete release of the lobe with an attempt at suture of the everted margins of the opening, and the third was a free tissue graft. A mass of fat was removed from the buttock about 1½ inches long and 1½ inches in the other diameters, was laid into the cavity, and was sutured in place by chromic sutures passed through it and the adjacent pulmonary parenchyma. Two of these sutures traversed the interlobar fissure to grasp the adjacent normal upper lobe. The fibrous adhesions between the lower lobe and the parietal pleura were also employed for the anchorage of some of the sutures.

After operation the pneumothorax recurred partly and the wound was opened at one angle in order to permit some of the air to escape. One week after operation the roentgen film demonstrated a subtotal pneumothorax. Subsequent films showed the complete disappearance of the pneumothorax. The case was closed at follow-up on April 7, 1937. The existence of the pneumothorax in the early stage after operation could be attributed to failure of the graft, but it appears more likely to have been due to leakage of air between bronchial fistulas and graft. This has occurred in other cases in which healing has taken place, as will be noted below.

CASE 7.—B. J., No. 392056, female, twenty-four years old, Mount Sinai Hospital, was operated upon for a large chronic putrid abscess of the lung in 1930. Many thoracoplastic procedures were performed in the hope of obliterating the large lattice lung which persisted. They failed to appreciably reduce the size of the lesion. Subsequently, a muscle flap was turned into the cavity. This procedure also failed. A fat graft was decided upon in April, 1936, although it appeared unlikely that it would succeed because a purulent discharge continued from the cavity even when frequent dressings were employed. The fat mass, approximately 4 inches in each diameter, was divided into several sections and placed into the large cavity. The cutaneous margins were freed and sutured.

The wound remained healed, or rather apparently healed, for a period of three weeks. Purulent discharge then began. The wound was opened and the necrotic fat masses were removed.

There were no undesirable consequences as the result of the failure of the fat graft in this case.

CASE 8.—S. M., No. 392108, male, thirty-eight years old, Mount Sinai Hospital, was operated upon April 21, 1936, for an acute putrid abscess of the left upper lobe. The lesion was a dumbbell-shaped one of considerable size with surprisingly stiff walls for an abscess of three or four weeks' duration. The cavity was clean within two weeks and the patient was quite symptom free. However, the walls of the cavity remained stiff. The wound was closely observed for the succeeding two weeks. There was no evidence of reduction in size. The stiffness of the walls persisted as well as the patency of several bronchial fistulas.

Although spontaneous healing was considered a possibility or even a probability, the impression was gained that repair would be a slow and tedious process. Accordingly, a fat graft was decided upon, primarily for the purpose of shortening the convalescence. It was performed on May 18, 1936. A fat cylinder about 2

inches long and ½ inch in the other diameters fitted snugly into the bilocular cavity and blocked the fistulas. Because of the depth of the cavity (pectoralis major had been divided at the primary operation for the purpose of access to the abscess), an additional mass of fat was placed over the one in the cavity. The musculature and skin were then closed in separate layers.

There was a slight bronchial blow for the first ten days after operation, and there was also a superficial transient infection of the wound. Small quantities of blood-tinged sputum were expectorated in the early postoperative period. This has also been noted in some of the other cases. Otherwise the course was uneventful. The wound has remained healed, the roentgen film at follow-up on Sept. 16, 1936, indicated normal pulmonary fields. In October, 1936, a bronchogram demonstrated the existence of some contraction of the bronchi of the upper lobe in the region in which the abscess was situated, but was normal otherwise. The patient has remained well.

CASE 9.—L. K., No. 397543, female, thirty-four years old, Mount Sinai Hospital, was operated upon March 27, 1936, for long-standing pulmonary tuberculosis with cavitation in the upper lobe. A thoracoplasty was performed as well as an effort at cavernolysis. The operation had no appreciable effect upon the pulmonary cavity and the sputum remained positive. About two months later (May 20, 1936) the tuberculous cavity was approached through pleural adhesions, was entered, two or three drams of pus were evacuated, and the roof was partly excised. Large bronchial fistulas were noted. The sputum became negative shortly after operation.

In order to attempt to avoid a permanent communication between the tuberculous cavity and the skin, a fat graft was decided upon as soon as the cavity appeared healthy enough to receive it. The fat transplantation was performed on June 2, that is, less than two weeks after the tuberculous cavity had been opened. The fat mass measured about 1½ inches in each diameter and was divided into three portions which approximately fitted the abscess cavity. The edges of the skin were strapped.

The wound healed and has remained healed. Upon a subsequent occasion a sinus in the soft parts, and not in the region of the cavity, appeared. It was found to lead to a superficial dead space and was opened. The wound healed soon thereafter. The sputum has remained very scanty and consistently free from tubercle bacilli. The last examination was made in the early part of 1937.

CASE 10.—S. W., No. 394118, male, twelve years old, Mount Sinai Hospital, was operated upon for an enormous, essentially unilocular nonputrid abscess in the right lower lobe on April 16, 1936. A fully developed lattice lung with all the characteristic features was noted at the time of evacuation of the abscess. There was rapid improvement in the clinical manifestations after operation. The cavity in the lung reduced somewhat in size and showed a distinct tendency toward healing. Accordingly, the skin was permitted to close. The roentgen films, however, demonstrated the persistence of the cavity and a fat graft for its obliteration was therefore decided upon.

The operation was performed on June 4, 1936. The cavity was entered and found to be clean. The patient was a thin subject and all the fat that was needed for the purpose of the complete filling of the cavity could therefore not be obtained. A fat mass 3 inches long and 1½ inches in the other diameters was dissected out and divided into several portions which were placed within the cavity. The skin was approximated by strapping after it had been mobilized.

The postoperative course was uneventful. A roentgen film demonstrated the existence of a fluid level, which was presumably due to the fact that the cavity was not completely filled by the fat graft. There was a slight discharge from the

wound for a short period of time. Within a few weeks of the time of operation, the fluid level had disappeared and the wound was healed. The patient has remained entirely symptom free.

CASE 11.—R. R., No. 393421, female, eleven years old, Mount Sinai Hospital. Three months before admission, several operations were performed for pyopneumothorax. Fever continued and the first step was the establishment of adequate drainage. This was performed shortly after admission on May 19, 1936. At that operation two hemispherical pulmonary cavities opening into the pleural cavity were noted. The external orifices on the visceral pleura were from ½ inch to ¾ inch in each diameter. The cavities, however, were appreciably larger. In order to reduce the size of the pleural dead space a phrenic severance was carried out on June 2. The cavities with their bronchial fistulas persisted. In an effort to close them, fat implantation was performed on June 10, 1936. In this case the fat grafts were divided into multiple minute fragments. Each of the pulmonary cavities was filled with these fragments to the level of the visceral pleural surface. It was hoped that blood clot or serum would hold them in position because they could not be sutured in place.

A bronchial blow continued from both cavities after operation. In the case of one of the cavities all of the fat grafts were expelled by coughing. In the case of the other cavity, some of the grafts appeared to have been retained and early closure of the bronchial fistula occurred together with the closure of the cavity. The case, therefore, is one in which either complete or partial failure occurred.

CASE 12.—M. L., No. 393787, female, thirty-nine years old, Mount Sinai Hospital. The lesion was a tuberculous cavity in the lung of seven years' duration. The Wassermann test was positive upon several occasions, and antiluetic treatment had been administered. It seemed most unlikely that a thoracoplasty would influence the size or shape of this ancient lesion. Accordingly, drainage was decided upon. This was performed through agglutinating pleural adhesions. Sputum became negative after operation and the open cavity fairly clean. Accordingly, a fat transplantation was decided upon. It was performed on July 10, 1936. The fat mass, about 1 inch in each diameter, was divided into several fragments. The skin margins were freed and strapped, with drains placed down to the soft parts.

An indolent infection occurred and the grafts were expelled two weeks after operation.

CASE 13.—R. B., No. 399494, male, was five years old on his first admission in 1932, Mount Sinai Hospital. A year and a half before his admission a pyopneumothorax had been drained. A chronic bronchopulmonopleural fistula persisted. At an exploratory thoracotomy a typical lattice lung occupying much of the right lower lobe was noted. The decision was then made to attempt obliteration of the pleural space by thoracoplasty. A number of thoracoplastic procedures were carried out in 1932 and 1933. They did not materially influence the lesion. The lattice lung persisted and a moderate discharge came from its depths.

In March, 1935, a very difficult and partial lobectomy was performed. This eradicated much, but not all, of the lesion. A moderate discharge continued with the persistence of several bronchial fistulas. The wound was revised upon two subsequent occasions. Finally, on Nov. 20, 1936, the decision was made to attempt to fill the residual lattice lung with a fat graft. The patient was a very thin subject and the largest segment of fat which could be removed measured no more than an inch in each diameter. It occupied only part of the cavity and for this reason was held in place by a small gauze pack. The skin margins were approximated by strapping.

For a few days after operation there was a slight fistulous blow. Healing occurred promptly after the removal of the pack. The wound has remained healed, and the patient has been entirely symptom free since operation. When last seen at a recent date, he had gained greatly in weight and strength and was well.

CASE 14.—W. M., No. 386454, male, twenty-three years old, Mount Sinai Hospital, had been operated upon repeatedly for empyema since 1931. When admitted in September, 1935, fever had been present for many weeks. At operation, shortly after admission, an extensive empyema was encountered. It was widely drained. The cause of the empyema was only realized several weeks after operation. At that time a bronchial fistula made its appearance. It was explored and a bilocular pulmonary abscess was encountered at the site of the bronchial fistula. The abscess cavity was exceedingly stiff walled and presented on its floor the orifices of large or dilated bronchi.

Although the empyema cavity became clean and reduced in size progressively, there was no evidence of healing of the chronic pulmonary cavity. It appeared to be a simple one for closure by a musculocutaneous flap, and this procedure was carried out on Nov. 28, 1935. The operation failed and the cavity persisted. A continuous infection also persisted, which was undoubtedly the cause of the failure of the flap operation. Radiotherapy reduced appreciably the amount of purulent discharge.

A fat graft was finally decided upon. It was performed on Nov. 23, 1936. The bronchopulmonary cavity was not large, and the size of the graft was about 1 inch to 1½ inches in each diameter. The skin margins were freed and approximated. Primary union took place and the wound has remained healed.

CASE 15.—C. G., No. 399657, female, now eleven years old, Mount Sinai Hospital. In 1934 multiple closed drainages were performed for separately encapsulated pyopneumothoraces. Open operation was performed at a later date, and subsequently a number of revisions were carried out. By the end of 1935 a fully developed lattice lung existed. This was drained partly by removal of overlying rib in the early part of 1936. Rather profuse discharge from the wound continued, and this was treated by radiotherapy with some improvement. In October and again in November of 1936 the cavity was unroofed in the hope that discharge would become less. There was no great improvement and the field was obviously unfavorable for a fat graft. Nevertheless, this was performed on Dec. 8, 1936. The cavity was spherical, and about 2 inches in each diameter, when the child coughed or strained. Its lining was smooth and there were a series of trabeculations with at least a dozen bronchial fistulas of varying size between the trabeculae. A fat mass of appropriate size was removed from the buttock and divided into a number of separate portions. These were placed into the various recesses and the central cavity and came up to the rib level. After mobilization, the soft parts and skin were closed.

The postoperative course was at first uneventful. About two weeks after operation there was some fever. The wound was probed, and one to two drams of a mucoid purulent fluid escaped. There was a small fistula for a time and the roentgen film of a fluid level in the cavity. Failure was anticipated. However, complete healing occurred and the wound has remained healed. In the early phases there was slight mucopurulent expectoration. The child is well.

CASE 16.—S. E., No. 400544, male, forty-nine years old, Mount Sinai Hospital, was operated upon for an acute putrid abscess in the paravertebral portion of the right upper lobe on Nov. 4, 1936. The postoperative course was not satisfactory, and a more deeply placed adjacent putrid pulmonary abscess was opened on Nov. 20. There remained a large cavity which rapidly became clean and which was multilocular with a number of bronchial communications. The progress of the wound was care-

fully observed after operation, and there appeared to develop a tendency toward increasing stiffness of the walls of the cavity. At any rate, there was apparently no tendency toward healing. The impression was gained that the wound would probably heal in the course of time, but that it would be a long process. A fat graft was therefore decided upon.

The transplantation of fat was carried out on Dec. 11, that is, three weeks after the second pulmonary abscess had been opened, and five weeks after the primary one had been unroofed. The pulmonary cavity, which was roughly cylindrical, was about 3 inches long and 2 to 2½ inches in the other diameters. A mass of fat of this size was removed from the buttock and was divided into sections. The separate fragments were put in place and made a form of mosaic filling of the cavity to its superficial limits. The skin and musculature were mobilized and closed in layers. The postoperative course was entirely uneventful. The wound healed by primary union. The follow-up roentgenograms have indicated no abnormality. The cicatrix is flat. The patient was last seen at follow-up on May 4, 1937. He has remained well.

CASE 17.—A. S., No. 404124, male, thirteen years old, Mount Sinai Hospital, was admitted on February 1, 1937, with a history of six months' duration. At the onset the diagnosis of bronchopneumonia was made, and pus was obtained upon two diagnostic chest aspirations. It was removed by the Potain method. Fever, cough, and expectoration of purulent material continued. In the roentgen film on admission there was a homogeneous shadow with a fluid level. At operation, shortly after admission, an elastic mass in the substance of the right lower lobe was exposed. It was packed off and at the second stage one week later, it was entered. There was a fibrous shell of tissue overlying a cyst within the substance of the lung. The cyst contained purulent fluid which was evacuated. The collapsed cyst wall could then be removed without difficulty. After its removal there were noted a number of large and small bronchial fistulas.

The report on the cyst wall was infected echinococcus cyst. The pulmonary cavity soon became a clean bed with rigid walls. The bronchial fistulas persisted. The appearance was similar to that of lattice lung. The impression which was gained after several weeks' observation, was that the bronchopulmonary cavity, presumably present for some six months before operation, would probably persist indefinitely. Accordingly, a transplantation of fat was decided upon in the hope that healing might occur. It was performed on March 2, 1937, one month after the primary operation. The bronchopulmonary cavity was flat and saucerlike, 2½ inches by 2½ inches by 2 inches in diameters. A mass of fat of appropriate size was detached from the buttock and divided into small fragments. These were fitted into place. A gauze pack was placed over them to prevent expulsion, and the skin was mobilized and sutured without tension.

There developed an infection of the wound with some mucoid discharge. Three days after operation the wound was partly opened and several fat fragments escaped. However, only a small bronchial blow was present and this soon disappeared. On March 15 the wound was definitely healed. It has remained healed since that time, and the patient has been symptom free.

SUMMARY

The problem of operative eradication or closure of bronchopleural cavities (lattice lung) is sometimes a very difficult one.

A new procedure for the closure of bronchopulmonary cavities is described.

It consists of the free transplantation of fat into the cavity.

None of the criteria on which the successful free transplantation of any tissue (including fat) has been based, holds for the free transplantation of fat into bronchopulmonary cavities. The method was first employed empirically (and illogically) not because there was any expectation of a satisfactory result, but because the problem was one for which an adequate solution was not apparent. It was carried out in other cases because it succeeded in the first and not because a rationale has been evolved.

Some of the pulmonary cavities for which fat grafts have been employed were of recent origin, others of long duration. There was considerable variation in size and in antecedent cause.

The pathologic features of the pulmonary cavities were fairly uniform. They consisted in a smooth-lined, trabeculated wall, with bronchial orifices of varying size and number between trabeculae. Slight purulent discharge was present in some cases, more profuse discharge existed in other instances.

The simplicity of the operation and its freedom from danger when failure occurs is established. If indicated, the transplantation is carried out when the cavity appears to be as clean as can be anticipated.

The technique of operation consists in placing fragments of the fat graft into the different recesses of and into the main cavity, without any preparation of the bed. The skin (and soft parts, when available) should be approximated over the graft without any tension. A gauze pack for the retention of the graft is indicated in some cases.

The operation has been performed in 17 cases. There have been 3 failures, and 3 partial failures or partial successes. The operation has been completely successful in 11 cases in the sense of permanent closure of the open cavity. The fate of the fat graft is unknown, but absorption and replacement by fibrous connective tissue can be assumed. The mechanism of closure of the cavity is also unknown. The clinical results of the operation in all 11 cases are entirely satisfactory, regardless of the etiology, and problems often difficult or impossible of solution by other than extensive and dangerous operative procedures, have been solved by an exceedingly simple maneuver.

There have been several additional cases of fat transplant, all successful, since this paper was prepared for publication. In particular Case 7, a failure after fat graft, was subsequently subjected to operation with the idea of removing the cavity-bearing lower lobe. The latter could be but partially released. The result of this operation was a slight reduction in the size of the cavity. The fat graft was then implanted into the cavity and this time successfully.

DISCUSSION

DR. CARL EGGERS, New York, N. Y.—I am very much interested in Dr. Neuhof's presentation. I have tried to visualize what takes place when a free fat graft is transplanted into an old pulmonary cavity, for it must be remembered that such a cavity is lined with epithelium. Dr. Neuhof has some very nicely healed cases, which are symptom free. However, I wonder whether the implantation of fat had anything to do with their cure. I cannot conceive of a piece of fat put into an epithelialized cavity and actually playing a rôle in the obliteration of that

cavity, or of the bronchial fistulas which empty into it. It seems to me that the patients have been cured in spite of the fat transplant, not because of it. My impression is that other factors are responsible for healing, namely, sterility, or at least surgical cleanliness of the cavity, possibly mobilization of the surrounding lung, and approximation of the overlying tissues.

I have seen several cases of this type and have succeeded in healing them without the use of fat transplants. We have delayed operation until the wound was clean, the secretion small in amount and of a mucoid, rather than purulent character. Mobilization of the lung, at times with suture of the edges of the cavity, and approximation of the overlying tissues, have been relied on to lead to healing. I have never seen an epithelialized cavity of that kind filled with granulations; that is why they don't heal, because they are rigid and lined with epithelium. The secretion, which before closure is exuded externally, must go some place. It apparently rapidly diminishes in amount after external irritation ceases, and the little which forms is expelled via the bronchial tree.

Dr. Neuhof did not mention whether he had occasion to reopen any of these cases later in order to see what changes had taken place. I have had the good fortune to study one very interesting case of this type. The patient was a boy with a chronic lung abscess who went along for years before a drainage operation was done. A cavity the size of a walnut was found in his right lung, anteriorly. It was lined with epithelium and showed several bronchial openings. A fistula was maintained for several years, finally resulting in a pin point opening giving off a little mucoid secretion. Eventually he wanted it closed, and at operation we found that the cavity was still the same size as it originally had been. Mobilization of the lung with suture of the cavity margins and approximation of the overlying tissues has resulted in a permanent cure.

DR. EDWARD D. CHURCHILL, Boston, Mass.—In considering the transplantation of fat or muscle into cavities of the lung or pleura we must divorce the principles of tree surgery from the principles of thoracic surgery.

By virtue of its construction the lung is an organ designed for the success of transplantation of tissue to close cavities. My conception of the fate of these grafts is identical with Dr. Neuhof's idea, and based on the principles expounded by Halsted in a paper on the rôle of the blood clot in the healing of wounds. The lung being an elastic, flexible organ, all that is needed is a temporary filling which will plug the bronchus and become attached to the walls of the cavity. Then with its slow absorption, traction on the flexible adjacent portions of the lung brings the walls into approximation.

I do not hold the concept that the muscle or fat, or the fibrous tissue that replaces it, permanently fills the cavity in the sense that a dentist fills a tooth. For this reason, the criteria for the success of the operation are whether the walls of the cavity into which the graft is placed are flexible, and whether the bronchus is closed so as to encourage the absorption of air. The unpredictable and variable element determining failure or success is infection.

I am not surprised, therefore, at the success of the free fat transplantation reported by Dr. Neuhof as I visualize it as working exactly as a blood clot. In fact the exudate called forth may be as important as the graft itself. I am surprised at its success in as large a percentage of cases as reported by Dr. Neuhof, and I hope we shall have further confirmation as to the frequency with which it can be done.

DR. OWEN H. WANGENSTEEN, Minneapolis, Minn.—I have had no experience with the employment of a free graft of fat in the closure of pulmonary cavities, but I have some knowledge of the difficulties in closing the so-called "Gitterlunge"

or lattice lung. Any expedient which will accomplish this end as simply as Dr. Neuhof has described is worthy of trial.

Because of frequent failures with ordinary methods, Lebsche, while an assistant of Professor Sauerbruch at Munich, devised a method whereby the area of lung concerned was freed by sharp dissection, and the adjacent lung edges were sutured together over the bronchial stomata—a rather formidable procedure. Two years ago before this Association (J. THORACIC SURG. 5: 27, 1935) I reported two instances of lattice lung following spontaneous rupture of lung abscess into the pleural cavity, in which I oversutured a pedicled muscle flap over the multiple bronchial openings. That experience taught me that the bronchial openings of a lattice lung can be satisfactorily closed by this simple procedure. I also learned, however, that it was necessary to obliterate the residual empyema cavity, for despite great improvement attending cessation of drainage from the lung into the pleural cavity, the accumulation of fluid in the empyema cavity (the skin having closed) served to detach the muscle closure and caused the bronchial openings to leak again. I recounted then also, I believe, how a chronic duodenal fistula which had remained refractory to closure by other means became healed by the implantation of a pedicled muscle graft.

Professor Laxer, of Munich, has been the keenest exponent of the usefulness of the free fat graft. His use of it, I believe, has extended largely to noninfected cases. However, I do believe that successes have atended its use in the obliteration of residual osteomyelitic cavities—an occurrence which would lend support for the indication to which Dr. Neuhof has employed the free fat graft.

DR. HAROLD NEUHOF, New York, N. Y. (closing).—I remember Dr. Lilienthal's case very well, because it was a brilliant result which he obtained. The case was, however, one in which apicolysis was performed, as he has stated, and the large fat mass which was detached from the abdominal wall was employed for obliterating the endothoracic dead space. Had Dr. Lilienthal heard my presentation, he would have known that this dealt solely with the question of placing grafts within opened pulmonary cavities.

I think that Dr. Eggers is right concerning any form of graft "taking" within a completely epithelialized cavity. That, however, I do not envisage as being the case in any of the patients in the present series. Although in many of these cases there may be areas of epithelium here and there, much of the cavity is not lined with epithelium. I, for one, should not attempt any form of graft in a completely epithelialized cavity, unless I first tried to prepare that cavity by some form of denudation of epithelium.

From what Dr. Eggers had to say, I judge that it is his impression that pseudohealing took place in my cases, as in the case to which he referred. Pseudohealing did not occur in the cases which I have presented. They have been quite carefully followed up after operation for varying periods of time, up to three years, and bronchograms have been done in a number of instances. They have been x-rayed at reasonably frequent intervals. There are no remaining dead spaces and no cavities are to be seen in the later phases.

In one or two instances one notes in the bronchogram that the bronchi are more or less crowded together, but there is no obvious defect within the pulmonary parenchyma as far as one can ascertain. None of these patients has been reoperated upon, I am glad to say, so I really do not know what has happened to the grafts.

At the outset, I stated that it was my impression that this was a quite illogical procedure, and the only reason for advocating it is because it has worked. However, as Dr. Churchill has, I think, well pointed out, things happen within the pulmonary parenchyma, within the substance of the lung, that appear not to happen elsewhere

in the body. Certainly one of the things that is happening here is a tendency to what one might call a transient "take," which is quite surprising.

All these cases were infected to varying degrees. In most of them, the antecedent infection had largely subsided, but there was some degree of infection in all cases.

What has happened to that secretion which exuded from the bronchial mouths in part and partly from the surface of the lattice lung or pulmonary cavity, I do not know. I assume that as healing progressed, there has been a subsidence of the inflammatory process which has been present.

In the reading of case reports, you will note that some of these patients had for short periods of time expectoration of some purulent material, presumably from the region of the bronchi.

Dr. Churchill has suggested that the healing after grafting may be similar to that after blood clot filling of a cavity. That may be the case; as I say, I do not know. I do believe that the end-result, however, is some form of indifferent connective tissue, which, perhaps, in part draws together the walls of the cavity and permits some form of coaptation.

I think we all know that whereas the appearance of some of these lattice lungs strongly suggests a very extensive degree of destruction, in some instances, at any rate, one can by freeing the margins reduce appreciably the size of these lesions. The amount of destruction is perhaps more often apparent than real.

CARCINOMA OF THE THORACIC ESOPHAGUS

A Report of the Successful Removal in One Case*

Harold Brunn, M.D., and H. Brodie Stephens, M.D.
San Francisco, Calif.

CARCINOMA of the esophagus, surgically treated, has had very few successes to its credit. At the University of California Thoracic Surgery Clinic various measures have been used in the treatment of this disease. Surgery has been attempted unsuccessfully in many cases. As in other clinics, many of the cases were found to be inoperable after they were exposed, and nothing further was tried except palliative measures, such as x-ray. At other times operations were hopefully undertaken, but were followed by death immediately either from shock or some complication, or the patients recovered for a short time, from a few days to a few weeks, but without any permanent advantage. This has been the history of this operation.

We have presumed, therefore, to report this single case of the successful removal of a carcinoma of the thoracic esophagus, placed in a most difficult situation under the arch of the aorta. The report is made not so much because of any considerable modification in the technique advised by Torek[1] in his first case, but rather to emphasize its feasibility in a suitable case, and also because of certain difficulties which were overcome in this procedure and the method of handling them.

This subject cannot be discussed without giving credit to Torek for the first successful operation in 1913. His patient lived for thirteen years after operation and died of pneumonia at the age of eighty. There was no evidence of recurrence in this patient at the time of death. King[2] in 1936 states there have been but five cases of cancer of the thoracic esophagus treated successfully by surgical removal. The same author has two successful resections to his credit. His first patient was alive eleven months after operation, with no suggestion of recurrence at that time. King's technique differs but little from Torek's original description except for the introduction of pneumothorax preliminary to operation and dividing the operation into two stages. In the first stage the fourth, fifth, and sixth ribs are divided in the paravertebral line, and the vessels and nerves are ligated and severed. The incision is then closed and the second stage carried out a few days later.

Eggers[3] has reported several successful resections, the longest survival period in his group being eighteen months. He carries out the technique of Torek in resection of the thoracic esophagus.

---

*From the Division of Thoracic Surgery, Department of Surgery, University of California, San Francisco.

Read before the Twentieth Annual Meeting of the American Association for Thoracic Surgery, at Saranac Lake, N. Y., May 31, June 1, 2, 1937.

Grey Turner[4] has introduced his so-called "pull through" operation for the treatment of carcinoma in the region under discussion. This author frees the esophagus as high as possible through an abdominal incision, and then completes the separation through an incision in the neck. The esophagus is then pulled out through the abdominal incision. He reports a successful recovery by this method; the patient lived nineteen months after operation and died from nephritis. Autopsy revealed a small metastasis in the cardiac end of the stomach which apparently played no rôle in the causation of death.

Lilienthal[5] has removed a carcinoma of the thoracic esophagus through a posterior mediastinal approach and reformed the esophagus by means of a skin flap, thus restoring continuity of the swallowing tube. His patient lived one year and four months, and local metastases were present at the time of death.

Tudor Edwards and E. L. Lee[6] have recently reported a successful removal of the entire thoracic esophagus plus the cardiac end of the stomach. Their technique was similar to that of Torek. The type of anesthesia in this case is of interest. They employed spinal anesthesia and in addition gas and oxygen delivered through a mask. Their patient survived seven months and died of rather widespread metastases.

Our experience at the Thoracic Surgery Clinic of the University of California Medical School in the treatment of carcinoma of the thoracic esophagus has been quite similar to that of other clinics. Most of our patients are seen late in the course of their disease, and they are often too weak to recover even from the gastrostomy operation, or a careful examination at this time will reveal inoperable spread into the glands behind the cardia and along the lesser curvature of the stomach. We have made no serious attempts to dilate the area strictured by the tumor, since we hardly felt justified to run the risk of perforating such friable tissue.

There have been a few cases in which we have used the application of radium by means of threads connected at either end of the radium, the latter being drawn up into the esophagus through the gastrostomy opening. We have had no striking results from either deep roentgen ray treatment, using the 1,000,000 volt machine, or the direct application of radium. Nevertheless, the recent report by Crump and Kasabach,[7] in which the carcinoma had been completely destroyed after external and internal radiation, gives promise in the treatment of those cases considered inoperable. We believe Crump has developed an accurate and satisfactory method of intraesophageal radium application, and we are equipped to carry out the treatment as he has indicated.

The surgical treatment of carcinoma of the thoracic esophagus surely has not advanced in the twenty-four years since Torek's successful report. There have been no other five-year cures to our knowledge. We

are reporting a successful resection of the esophagus for cancer of the midportion, with the hope that these sufferers can be brought to the surgeon earlier, feeling the patient's chance of surviving the operation is fairly good, but recognizing the uselessness if metastases are already present at the time of operation.

A. R., a Mexican woman, fifty-four years of age, was admitted to the University of California Hospital on August 31, 1936, complaining of vomiting and inability to swallow solid foods, of two months' duration, a loss of 44 pounds in weight in the past year, and pain deep under the lower end of the sternum, appearing one month before the onset of the difficulty in swallowing. Her past history was irrelevant. Physical examination revealed a well-developed and as yet well-nourished female with no apparent evidence of weight loss. Except for marked dental caries, physical examination was negative. X-ray examination of the esophagus revealed an obstructing lesion at the junction of the upper and middle thirds. Esophagoscopy was performed on September 1, 1936. This examination revealed a normal upper third of the esophagus. Below this level the lumen became markedly narrowed and was replaced by granulation tissue. This tissue was very friable; it bled easily and cut with increased resistance. Microscopic examination of the biopsy material showed a neoplastic epithelial growth which had assumed a glandular pattern. Mitoses were frequent. The pathologic diagnosis was carcinoma of the esophagus.

On September 4, 1936, a Senn type of gastrostomy was performed under local anesthesia. No metastases were seen in the abdominal cavity. Between September 10 and September 15 a total of 1,200 c.c. of air was introduced into the left pleural cavity.

Esophagectomy was performed on September 19. Anesthesia was produced by 100 mg. of avertin per kilo, along with gas and oxygen, the latter being administered through an intratracheal catheter. The technique of Torek was followed. Almost the entire seventh rib was resected, as well as short segments of the sixth and fifth ribs in the paravertebral line. The pleural cavity was opened by an incision through the periosteal bed of the resected seventh rib. The esophagus was exposed in the usual manner by first incising the mediastinal pleura. The former was easily freed at its lower end and dissection was then carried upward. The tumor occupied the esophagus in the region behind the arch of the aorta and dissection here was difficult. A few hard, discrete lymph nodes were attached to the tumor. The tumor had slightly invaded the undersurface of the aortic arch and dissection was carried as close to this vessel as feasible. All bleeding encountered was satisfactorily controlled. Following the freeing of the tumor, the remainder of the upper esophagus shelled out easily. The lower end of the esophagus was divided aseptically with the scalpel between heavy silk ligatures. Both stumps were thoroughly carbolized, and the distal stump inverted into the stomach, using a double layer of purse-string sutures. In the course of the dissection some of the fibers of the left vagus were severed, but it was the operator's opinion that both main trunks were intact at the completion of the freeing procedure.

The patient was then turned on her back and an incision in the left side of the neck along the anterior border of the left sternocleidomastoid muscle was developed down into the superior mediastinum. The freed esophagus was pulled up through the neck incision and brought through the tunnel of skin and subcutaneous tissue as described by Torek. The tumor was then excised and the distal end of the upper esophagus anchored to the skin of the chest wall by several interrupted sutures of silk. During the dissection of the esophagus from its bed the right leaf of the mediastinal pleura had been ruptured, but had been temporarily plugged with a moist laparotomy tape. This rent now was satisfactorily closed with in-

terrupted sutures. The divided ends of the sixth and fifth ribs were approximated with chromic ligatures, and the exploratory incision in the chest wall was closed tightly. The pleural cavity was drained by a closed tube brought out through the ninth interspace in the posterior axillary line. A transfusion of 500 c.c. of citrated blood was given during the closure of the operative incision. The operation was completed in four and a half hours, and the patient was returned to the ward in remarkably good condition.

The postoperative convalescence was comparatively uneventful. The drainage catheter was removed twenty-four hours after operation because no drainage was present. Deep roentgen ray therapy was begun on the twenty-first postoperative day, and the patient left the hospital on the thirty-second postoperative day in good condition.

Microscopic examination of the resected esophagus revealed invasion of the entire wall of the esophagus by glandular epithelial cells, the tumor cells assuming a glandular pattern. The lymph nodes attached to the resected specimen showed invasion by the carcinoma.

Deep roentgen treatment was delivered to the region about the arch of the aorta between October 12 and November 25, 1936. Six portals were utilized, with a total dosage of 12,000 roentgen units.

Roentgen films of the chest taken three months after operation were negative for any evidence of metastasis. Repeat films of the chest were taken five months after operation because the patient was complaining of shortness of breath. Mottled densities, having a bronchial distribution, were now present in the bases of both lungs. These densities were interpreted to be the result of the intensive roentgen therapy administered four months before.

It is now eight and a half months after operation. The patient is at present strong and active. She is enjoying life and her dyspnea has greatly improved.

COMMENT

We wish to emphasize the importance of the pressure relations within the thorax to the successful outcome of any major intrathoracic operation. As our experience in thoracic surgery has increased, we have come to realize that the deaths[8] on the operating table and soon after surgery are due to circulatory failure, and the underlying cause of this failure is disturbed intrathoracic pressure. Preliminary pneumothorax, we believe, brings about a partial fixation of the mediastinum, thereby preparing the heart for the insult of a wide intrapleural exposure. With a partially fixed mediastinum there is much less paradoxical movement, and the closed side of the thorax can more easily handle its respiratory and circulatory functions. In addition, the heart has become accustomed, so to speak, to a one-sided shunt in the circulatory route, with the result that there is an absence of any circulatory shock at the time the thorax is opened.

Positive pressure intratracheal administration of the anesthetic gas is another aid in the successful completion of intrathoracic procedures

Introduction of oxygen under positive pressure is the only means of preserving life in the event of a bilateral open pneumothorax. We believe the necessary expansion of the contralateral lung can be much more satisfactorily accomplished through an intratracheal catheter rather than with the ordinary gas mask.

In the case reported, the right leaf of the mediastinal pleura was torn during the enucleation of the esophagus. No embarrassment of the heart or respiration occurred because of the satisfactory control of these functions by the positive pressure administration of the anesthetic mixture. Before the chest wall was closed, the right leaf of the mediastinal pleura was approximated with sutures, and before the intratracheal catheter was removed, all the air possible was aspirated from the contralateral thorax.

SUMMARY

A successful removal of the esophagus for carcinoma in its midportion is described. We believe the operation is made safer with the use of preoperative pneumothorax and the administration of the anesthetic under positive pressure through an intratracheal catheter.

Since there have been so many unsuccessful surgically treated cases and so very few successful results, a combination of circumstances would seem to be necessary to obtain a surgical recovery. For the present at least, this will include a patient who is in a good state of nutrition at the time of surgery, of a sturdy constitution that can weather a major operative procedure, and whose cardiovascular reserve is sufficient to meet the demands put upon it during the course of the operation. We believe our patient presented such qualifications.

REFERENCES

1. Torek, F.: The First Successful Resection of the Thoracic Portion of the Esophagus for Carcinoma, Surg., Gynec. & Obst. **16**: 614, 1913.
2. King, E. S. J.: Surgical Treatment of Carcinoma of the Thoracic Esophagus, M. J. Australia **1**: 399, 1936.
3. Eggers, C.: Resection of the Thoracic Portion of the Esophagus for Carcinoma, Arch. Surg. **10**: 361, 1925.
4. Turner, G. G.: Discussion on Recent Advances in Treatment of Cancer of the Esophagus From Surgical and Radiological Aspects, Proc. Roy. Soc. Med. **27**: 355, 1934.
5. Lilienthal, H.: Carcinoma of the Thoracic Esophagus, Extra Pleural Resection and Plastic, Ann. Surg. **74**: 259, 1921.
6. Edwards, A. T., and Lee, E. L.: Extirpation of the Esophagus, J. Laryng. & Otol. **51**: 281, 1936.
7. Crump, A., and Kasabach, H.: Report of "Proved Cured Case" of Squamous Cell Epithelioma Treated by Intraesophageal and External Irradiation, J. THORACIC SURG. **5**: 157, 1935.
8. Stephens, H. Brodie: A Consideration of Contralateral Pneumothorax as a Complication of Intrathoracic Operations, J. THORACIC SURG. **5**: 471, 1936.

TREATMENT OF CARCINOMA OF THE ESOPHAGUS BY MEANS OF SURGICAL DIATHERMY*

HERMAN J. MOERSCH, M.D.
ROCHESTER, MINN.

IN spite of the voluminous literature dealing with the problem of the surgical treatment of carcinoma of the esophagus, the end-results obtained by this method have been distinctly discouraging. The greatest obstacle to reaching a successful solution of this problem has been the factor of early diagnosis.

Early diagnosis is complicated by the fact that carcinoma of the esophagus is very insidious in its onset and is usually well advanced before it produces symptoms. To add to the difficulty the tumor is usually of a high degree of malignancy. That there is room, however, for progress in the field of diagnosis is apparent from a review of the case histories of 447 patients seen at the Mayo Clinic during a four-year period. The average duration of symptoms before the patient came under our observation was 7.1 months. It is, therefore, apparent that early diagnosis is the exception rather than the rule.

In dealing with large numbers of patients suffering from a disease such as carcinoma of the esophagus, one is bound by the laws of chance to encounter a case occasionally in which the disease is in a relatively early stage of development. The case I am about to present belongs to this group and illustrates what might be accomplished by the use of surgical diathermy.

REPORT OF A CASE

The patient, a man aged sixty-eight years, was admitted to the clinic on January 2, 1935, complaining of substernal pain. He had been under our observation some years previously because of nervousness and pruritus ani, from which he had completely recovered. His present difficulty had begun two months prior to his last admission, when he had first begun to suffer from a sense of burning under the lower end of the sternum which appeared with deglutition. He later described the sensation as if the esophagus was too narrow to permit the free passage of solid food. In spite of his statement that there had been no restriction in diet, he had lost 22 pounds (10 kg.) during this time.

Physical examination did not disclose anything of diagnostic importance, although the patient had obviously lost weight. This was found to have decreased from 163 to 141 pounds (from 74 to 64 kg.). The Wassermann test of the blood, urinalysis, a blood count, and roentgenologic examination of the thorax gave negative results. Fluoroscopic examination of the esophagus was reported as revealing a polypoid lesion in the upper third of the esophagus. Esophagoscopy was performed, with the patient under local anesthesia, and a polypoid growth was dis-

*From the Division of Medicine, the Mayo Clinic, Rochester, Minn.

Read before the Twentieth Annual Meeting of the American Association for Thoracic Surgery, at Saranac Lake, N. Y. May 31, June 1, 2, 1937.

covered 9½ inches (24 cm.) from the incisor teeth. The tumor appeared to originate from the right wall of the esophagus and almost completely filled the lumen. Tissue removed from the growth was examined microscopically and was reported to be squamous-cell carcinoma, grade 2, according to the Broders' classification (Fig. 1).

The patient was very insistent that an effort be made to remove the growth. Dr. Harrington and I discussed the problem with him, and it seemed to us that in view of his age and the location of the growth in relation to the arch of the aorta, an attempt to destroy it by means of surgical diathermy was a highly justifiable procedure.

Accordingly, on January 14, 1935, esophagoscopy was performed, with the patient under local anesthesia, and the tumor, which measured approximately 2.5 by 3 cm.,

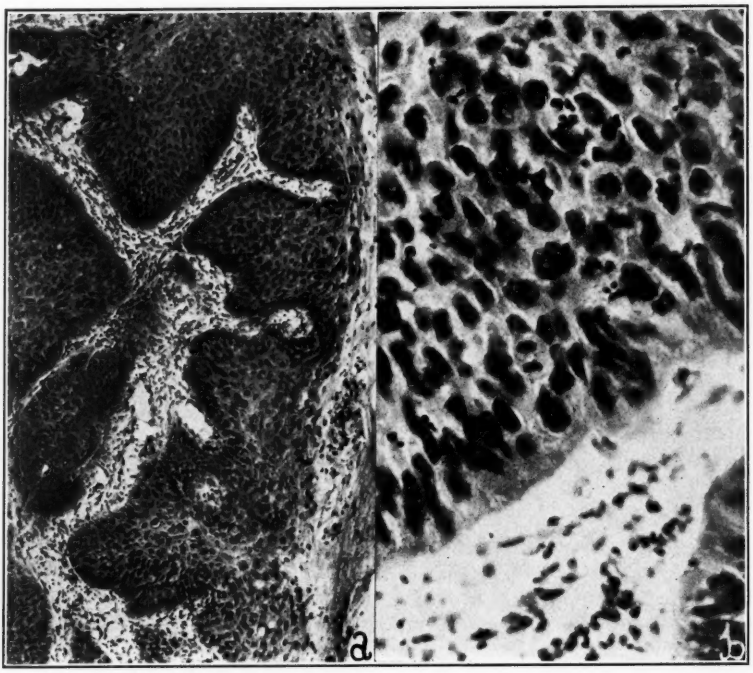


Fig. 1.—Section of tumor removed; *a*, low power; *b*, high power.

was destroyed by means of surgical diathermy, its point of origin from the esophagus being cauterized as thoroughly as possible. I was rather apprehensive lest coagulation produce a slough which might go through into the mediastinum. A small Rehfuss tube was inserted through the nose and into the stomach for feeding purposes. The following day the patient complained of substernal pain which was associated with an increase in the pulse rate up to 120 beats per minute, and in temperature up to 101° F. Within twenty-four hours, however, the temperature and pulse rate returned to normal and the pain disappeared. The Rehfuss tube was left in place for a week and was then removed; the patient was given a soft diet, but this was gradually increased in amount so that, by the end of the second week, he was able to eat a very liberal diet. Within a month he had gained 9 pounds

(4.1 kg.), and he was able to eat everything without difficulty. Roentgenoscopic examination of the esophagus at this time gave negative results. Esophagoscopic examination revealed three small areas of infiltration at the original site of the tumor. These were readily destroyed by coagulation. Following this, two courses of roentgen therapy were administered, at intervals of six weeks. The patient has remained well since and has undergone seven esophagoscopic and fluoroscopic examinations without any evidence whatsoever of a recurrence.

In conclusion, this case seemed worthy of reporting in view of the excellent results obtained. Further, it emphasizes the importance of early diagnosis and calls attention to the fact that the use of surgical diathermy may be of marked value in the surgical management of carcinoma of the esophagus.

DISCUSSION ON "CARCINOMA OF THE ESOPHAGUS," BY DR. BRUNN AND DR. STEPHENS; "SUCCESSFUL TREATMENT OF CARCINOMA OF THE ESOPHAGUS BY MEANS OF SURGICAL DIATHERMY," BY DR. HERMAN J. MOERSCH

DR. HOWARD LILIENTHAL, New York, N. Y.—I should like to call attention to one or two important points: First, that in spite of Torek's successful case, the other case referred to today, and a few others that have been reported, the disadvantage of entering the pleural cavity, except temporarily, or accidentally, I think is very great. I don't wish to take up your time by too many arguments, but would call your attention to the case that was reported in 1920, or thereabouts, in which I removed a carcinoma of the esophagus by the posterior mediastinal route, and without gastrostomy. The patient could swallow, and he lived for about a year and a half, finally dying of recurrence. For some months he apparently was perfectly well. The actual cause of death in that case was the use of radium, which then was not particularly well understood and which burned an opening between the esophagus and the bronchus. The important point that I wish to make is the mediastinal approach. If you will try the mediastinal operation in a cadaver, you will see how beautiful the exposure is. You will see that you do not have to interfere with any of the important vascular structures in the chest, that you have the opportunity to do an operation which I have tried a few times but with only temporary success. The ideal operation is esophagogastrostomy as a first stage.

If you make a large opening in the diaphragm, you can pull the stomach up to the second rib, or even further, without any particular shock or trouble to the patient. You then cut off the esophagus above the tumor and implant it into the stomach through a gastric incision, which is a very simple procedure. Those of you who are interested will find it in a book of mine.* Strangely enough, Sauerbruch had the same idea at approximately the same time. Mine appeared first, but his probably was done at about the same time. It was during the war and we weren't in very close contact.

The idea is, then, to perform an esophagogastrostomy through the mediastinum. Don't be afraid of the mediastinum. Nothing can happen if this space is sufficiently opened. Even if it becomes infected, nothing bad will happen. After you do an esophagogastrostomy high up, then you simply ligate the lower part of the esophagus which contains the growth and drop it back, covering it with a piece of rubber dam or gauze for a second stage. In four or five days after the operation of esophagogastrostomy, union will be complete and the patient will be able to swallow soft solids. I have proved that with one of my cases.

*Thoracic Surgery, W. B. Saunders, Philadelphia, 1925.

The next thing to do is to deal with the cancer itself, which is now entirely free. One may excise the cancer and drop the lower esophagus stump back through the cardia.

The great advantage is there is no external tube after these operations. The external tube to me, in spite of Dr. Torek's case and this one you just heard about, would seem a very disagreeable sort of thing to carry about with one. With esophagogastrostomy, you have accomplished a lot more for the patient's comfort.

I did not hear the Doctor say whether these patients received previous pneumothorax so as to relieve—for the time of the two, three, or four hours of the operation, the strain on the heart and the sudden change in the circulation, but I think it would be a wise procedure. I know I would do it if I had another case.

About the mediastinal infection, it has been my custom in cases of very large lung abscess to open them through the mediastinum and not enter the pleura at all. You have heard enough during this meeting to know that it is almost impossible to avoid having an infection of the pleura with a very poisonous kind of empyema.

I only mention this now to show you how simple it is to go in through the mediastinum, not only in the cases of the esophagus operation, but in the event of a large lung abscess. I have presented one or two of those cases; I think they are published in this Journal. At any rate, it saves a tremendous amount of shock and provides drainage from the upper or lower part. The pleura will not become infected. This is only mentioned here to show you that going in through the mediastinum is not a dreadful thing at all.

One word about the electrocoagulation. I am a great believer in electrosurgery. I do most of my operations by electrosurgery, including the skin incision, especially in thoracoplasty. I have taken out ten ribs in thoracoplasty without a single ligature and have obtained primary union, so you see I am "sold" on electrosurgery.

DR. CARL EGGERS, New York, N. Y.—I don't think one should let an opportunity go by to discuss a case of cancer of the esophagus, which has been cured, at least temporarily. Those surgeons who have continued to do pioneering work in the treatment of carcinoma of the esophagus in the face of great difficulties know what a pleasure and satisfaction it is to occasionally have a case recover. I, therefore, want to congratulate Dr. Moersch and Dr. Stephens on their brilliant results.

It seems to me that the most important thing at the present time is to establish confidence among the medical profession that this lesion is amenable to treatment. Whether radiation or surgery is used, it is important that treatment be instituted early. Both Dr. Moersch and Dr. Stephens referred to the difficulty of early diagnosis, and stated that it is usually six to seven months after symptoms are first noted before patients present themselves for treatment. When one considers that the first symptom is usually some difficulty with swallowing, and that this means advanced disease, it becomes apparent how important it is to refer patients for treatment at the first sign of difficulty. One should not delay and try one thing after another, knowing that conservative treatment is not curative, but one should be guided by the knowledge that only complete destructiion or removal of the lesion may bring about a cure. Dr. Torek's operation is the one which has had more operative successes than any other, and it has, therefore, helped more to encourage surgeons to continue this type of work, than any other procedures. At a recent combined meeting of the New York Surgical Society and the Philadelphia Academy of Surgery Dr. Garlock showed two cases cured by that method, and now come Dr. Brunn and Dr. Stephens with another. It is certainly gratifying to hear of this.

The operation is a formidable one and is followed by a rather high mortality which may be due to shock, infection, or other complications. In order to reduce

the incidence of these, we have tried to devise a practical method of dividing the operation into stages. Instead of doing the major part of the intrathoracic work first, then turning the patient over for the operative procedure on the neck and finally turning him back again to complete the intrathoracic work, we have lately divided it into two stages. After a preliminary gastrostomy we begin at the neck, making an incision similar to the one used for exposure of an esophagus diverticulum. One is able to work down on the esophagus very nicely and bluntly free it out of its bed almost down to the arch of the aorta. With the aid of gentle traction a heavy silk ligature may be placed well down, a clamp applied above it, and the esophagus then divided with the cautery. The lower stump is allowed to recede into the mediastinum, while the upper end is brought out at the neck and implanted as in Dr. Torek's operation. The operation isn't shocking at all, and the patient may be out of bed on the following day. As soon as the wound has healed, the thoracic part of the operation for removal of the tumor may be done.

So far I have not had a successful case with this method on account of extensive involvement, but it promises to be helpful. In addition to dividing the operation into two stages, the procedure has the additional advantage that it permits the patient to eat normally afterward. Whether one succeeds in removing the tumor or not, a rubber esophagus may be inserted between the esophagus stump and the gastrostomy, thus permitting the patient the pleasure and satisfaction of masticating and swallowing food.

The same method lends itself to extirpation of a carcinoma affecting the upper third of the esophagus. I recently did a resection of a carcinoma extending from the hypopharynx downward for at least three inches. It was possible to free the esophagus well below the tumor and to ligate and divide it, and then to bring the tumor out at the neck. It was removed at a later period, after adhesions had formed to protect the mediastinum. The patient made a good recovery.

Dr. Moersch and Dr. Stephens deserve our thanks for bringing this subject up for discussion, and by the presentation of two successful cases stimulate our interest.

DR. ETHAN FLAGG BUTLER, Ithaca, N. Y.—First, a word of congratulation to Dr. Stephens and Dr. Moersch—next, a word as to the surgical approach to the thoracic esophagus. The extrafascial apicolytic thoracoplasty described by Semb is virtually an upper posterior mediastinotomy. It affords ready access to the upper thoracic and lower cervical portions of the esophagus. It should afford an easy pathway to the anterior surface of the neck. The pleural cavity would not have to be opened. It is an approach that should be borne in mind when dealing with lesions situated in the upper half of the thoracic esophagus.

DR. HERMAN J. MOERSCH, Rochester, Minn. (closing).—It is only fitting that I should take this opportunity to pay tribute to the patient's home physician in this case, as it was only through his diagnostic acumen that the carcinoma of the esophagus was recognized at such an early stage.

Dr. Eggers' observation on the necessity of trying to impress on the medical profession that if cancer of the esophagus is seen in an early stage something may be accomplished from a therapeutic standpoint is very significant. The teaching of Dr. Jackson should be constantly kept in mind that whenever a patient presents symptoms referable to the esophagus, immediate esophagoscopy is indicated, as it is only through such means that carcinoma of the esophagus may be recognized at an early stage.

A NEW CONCEPTION OF TUBERCULOUS EMPYEMATA BASED ON THEIR PATHOLOGIC PHYSIOLOGY*

Importance of Bronchial Fistulae in Their Prognosis and Management; Significance of Gas Analysis of the Pneumothorax Air

Pol. N. Coryllos, M.D., F.A.C.S.
New York, N. Y.

Part I. Theoretical Considerations

THIS study is limited to purulent exudates complicating pneumothorax. Straw-colored serous effusions which accompany 50 to 70 per cent of all pneumothoraces are not included in this paper. However, as often it is difficult to differentiate between these effusions and "turbid and puslike" exudates, the latter will be placed with empyemata.

Today tuberculous empyema represents one of the most distressing complications of pneumothorax and one of the most confused problems in pulmonary tuberculosis. Very little definite and clear information is available on its pathogenesis, pathologic physiology, and treatment, notwithstanding the voluminous literature upon this subject. In fact, while we possess positive knowledge of the histology, pathology, and bacteriology of these exudates, we know very little, if anything, about the mechanism of their production and the factors which determine their course, which sometimes is smooth and benign while at other times is extremely septic and rapidly lethal. Neither do we know why tuberculous empyema may gradually improve and become quiescent for months or years and then again become toxic. We know even less of any reliable methods allowing us to predict, with any degree of accuracy, the future course of these exudates, or to prevent their toxic evolution. Differences of opinions wax even greater when we come to their treatment. This question is of paramount clinical importance when we consider that the mortality of mixed infection empyemata still remains around 70 to 80 per cent.

A study of these empyemata carried on for the past five years in Sea View and Metropolitan Hospitals of the City of New York, led us to new conceptions of the pathogenesis, prevention and treatment of this complication. Impressed by the constant presence of bronchial fistulae in empyemata operated upon by the Schede procedure and in all toxic postpneumothorax empyemata, which came to autopsy, we have endeavored to investigate further the pathologic physiology of

*From the Departments of Thoracic Surgery of Sea View and Metropolitan Hospitals of New York and the Laboratory for Surgical Research of Cornell Medical College.

these bronchial fistulae in a considerable number of cases of pneumothorax complicated or not by pleural fluid. For that purpose we used the method which we had described in a number of papers,[1, 2, 3, 4, 5] namely, systematic *gas analysis of the air contained in the pleural cavity.* This method has proved to be sufficiently sensitive and accurate as to render easy the so far uncertain diagnosis of bronchial fistulae. Furthermore, it enabled us to detect them even when the usual clinical symptoms and laboratory signs were absent, and to formulate the prognosis in each individual case with a high degree of accuracy. The data obtained by this method have constantly been in agreement with parallel changes in pleural pressures, and cytologic and bacteriologic modifications of the pleural fluid.

Thus, we gradually came to reconsider prevailing conceptions of the pathogenesis and management of tuberculous postpneumothorax empyemata, and arrived at conclusions radically different from the generally accepted ideas. If these conclusions prove to be correct, a great step could be realized in the prevention and treatment of this distressing morbid syndrome.

Because of the importance of the subject, we deemed it advisable to divide this study into three parts. In the first part we reviewed the actual knowledge of these empyemata. In the second part, these conceptions were discussed in the light of our recent findings. In the third part, our conception and its practical applications were presented.

I ACTUAL KNOWLEDGE OF PATHOGENESIS OF TUBERCULOUS EMPYEMA COMPLICATING PNEUMOTHORAX

Before the era of pneumothorax, pleural effusions and empyemata developing in tuberculous patients were not considered as very serious complications. In fact, in the classic monograph of Grancher and Hutinel[6] (1887), Laënnec,[7] Herard and Cornil,[8] Lendst[9] and others were quoted as believing that frequently simple tuberculous pleural exudates, or even purulent tuberculous pleurisies, had a rather beneficial influence upon the evolution of pulmonary tuberculosis. However, the prognosis was considered as incomparably more serious when spontaneous pneumothorax had preceded tuberculous pleurisy. In these cases, according to these authors, pleural effusions were almost invariably changed into purulent exudates with septic manifestations and death.

According to the older pathologists, pleural effusions were, as a rule, preceded by "dry pleurisies." The initial changes, according to Grancher and Hutinel,[6] "are observed in the visceral pleura. A thin layer of fibrinous exudate containing endothelial and white cells covers the visceral pleura, slightly thickened at that place. *Tubercles are always found in the periphery of the lung at the site of this dry pleurisy.*

LIBRARY
BOSTON UNIVERSITY
SCHOOL OF MEDICINE

Similar lesions develop on the visceral pleura. The epithelium covering both pleurae gradually disappears, and fusion of the two serosas, parietal and visceral, takes place. These pleurisies are due to the extension of the pulmonary disease to the visceral pleura. They are, therefore, more frequently localized at the apices and explain the great frequency of apical adhesions." Louis[10] in 112 autopsies found only one case free of adhesions. Frequently in chronic tuberculosis complete adhesions of the two pleural layers were found at autopsy. Besides these "dry pleurisies," Grancher and Hutinel[6] described "pleurisies with exudate," of which they described two separate forms. One, "exudative, serous and occasionally serosanguineous, with congestion of the capillaries of the visceral pleura, and with very few or no tubercle bacilli." This form had a benign evolution as long as the fluid remained serous and straw-colored, which as a rule was absorbed.

The second form comprised the group of "purulent tuberculous pleurisies." In these cases the pleural lesions were different. Instead of vascular congestion, there was obliteration of the capillaries. The visceral pleura instead of being covered with vascular granulating tissue, as in the previous form, presented patches in which the vessels were obliterated and the thickened serosa was infiltrated with tubercles, either caseated or on their way to caseation. *Often these patches were covered by necrotic tissue underneath which a tuberculous ulceration was present. These ulcerations varied in their size and depth. They could be very small, almost punctiform, and superficial. At other times they were deep and created communications between the pleural cavity and small bronchioles, or even larger bronchi.* Their evolution was the same as of all tuberculous abscesses. They ulcerated in their deep layers while there was a continuous tendency to close at the periphery. Grancher and Hutinel,[6] therefore, concluded "the differences between serous and purulent tuberculous pleurisies depend upon the nature of the lesions present on the surface of the lung," and they add, "most probably also on the additional pyogenic infection, always possible *when pleuropulmonary fistulae are present.*"

In this monograph of Grancher and Hutinel,[6] there is naturally no mention of artificial pneumothorax. However, there is a detailed description of the pathology of the production of spontaneous pneumothorax. "This complication," they say, "is very rare in old cases of pulmonary tuberculosis (1 in 100 cases). On the contrary, it is more frequent in recent cases. In 46 cases studied by Weil,[11] 40 occurred in the first four months of the disease. In the recent cases," they add, "where no protective adhesions had developed, caseation of subpleural tuberculous lesions may cause formation of pleuropulmonary fistulae. Because of the negative intrapleural pressure, air rushes into the pleural cavity, separating the two layers of the pleura

and causing the collapse of the lung. At the same time, purulent exudate develops containing a great number of tubercle bacilli and often pyogenic microbes."

We quoted rather extensively this old work of Grancher and Hutinel[6] because it represents the conceptions of the phthisiologists upon this subject before the eras of artificial pneumothorax and of x-rays. We wanted also to point out that the pathology of the tuberculous effusions was perfectly known at that remote time, and that very little, if anything, has been added since then. In fact Jaffé[12] in a recent article (1935) expresses exactly the same ideas. "The lack of anatomic and histologic specificity," he says, "is particularly striking in the purely serous effusions, which carry the earmarks of a massive perifocal exudation in highly sensitive allergic individuals. In many cases tubercle bacilli can be discovered in the fluid by animal inoculation, but also it may prove sterile." The same author says, "the most severe form of exudative tuberculous pleuritis is the empyema. It follows *the perforation of a liquefying caseous lesion of the lung.* When a tuberculous lesion breaks into the pleural sac, secondary invaders, especially streptococci, are often present in the pus and the rôle of these secondary invaders is very important."

An interesting and very elaborate monograph was recently published (1935) on "Para-Pneumothoracic Empyemata," by Monaldi,[13] of the Institute of Carlo Forlanini in Rome. In this monograph, Monaldi recognizes the importance of *bronchial fistulae* in the production of empyemata, although he considers them of lesser significance. "The so-called latent perforations," he says, "which are so named because *they cannot be elicited by any clinical method at our disposal,* may be the cause of empyema with acute onset. . . . It is possible that they may be more frequent than is believed. . . . These perforations may be either small, superficial erosions of the lung parenchyma, or may be real ulcerations of the diseased parenchyma, leading to a pleuropulmonary fistula (Bard). However," adds Monaldi, "Morelli[14, 15] affirms that empyema may develop without bronchopulmonary fistula." Monaldi[13] shares that opinion, as is shown by his involved classification of tuberculous empyemata which we believe of interest to reproduce here.

I. *Simple Tuberculous Empyema*

A. With acute onset (acute toxemia) 1. Pleural perforation 2. Following tuberculosis of the pleura	a. Partial nonefficient pnx. b. Efficient pnx.
B. Chronic (chronic toxemia): changed into empyema following: 1. Pleural exudate treated with pnx. 2. Simple effusion of pnx. 3. Acute empyema of pnx. 4. Exacerbation of encapsulated empyema.	c. Efficient pnx.; or at the end of pnx.
C. Encapsulated empyema (atoxic) (abandoned pnx.)	d. Bilateral simultaneous pnx.

II. *TBC. Simple Infected Empyema*	
A. With transient infection	
B. With permanent infection	
III. *Associated Septic Empyema*	
A. Acute. 1. Closed (primary-secondary)	1. With pulmonary lesion cured
B. Chronic. 2. Open (parietal fistula) (pleuro-pulmonary fistula)	2. With persistent pulmonary lesions

This classification, which is one of the most complete ever produced, gives a good idea of the state of confusion in the conceptions prevailing today on tuberculous empyemata.

For Monaldi the most important etiologic factors in the production of these exudates are mechanical. He quotes the following: First the special mechanical conditions of the pleura, the two layers of which constantly tend to retract one from the other because of the elasticity of the lung and to form a real pleural space. Thus a constant suction on the pleural vessels is created which favors the production of effusions. This factor would be helped by the fact that in tuberculosis, exudative processes are much more active than the proliferative ones in the organs which, as the pleura, present free surfaces and tenuous and reticular texture.

A second factor favoring the development of pleural exudates, says Monaldi, "is the variations of pressures in the pleural cavity during the respiratory cycle, because pleural negative pressures exert an important influence upon the permeability of the pleural vessels." (Monaldi, Bottari and Bobolini.) Therefore, Monaldi[13] believes that in recent pneumothoraces where the respiratory oscillations of intrapleural pressures are most marked, the production of pleural fluid is most rapid. On the contrary, he says, in old empyemata in which the immobilized chest and lung move little and the intrapleural pressures present only slight oscillations, the formation of fluid is slow and rare. Likewise in pneumothoraces in which pressures have been maintained constantly positive, and in partial and encapsulated empyema with rigid walls, there is but slight tendency for rapid production of fluid. The favorable action of phrenicectomy in empyemata would be due, according to Monaldi, to the decrease of variations of intrapleural pressures. This conception, says Monaldi, is corroborated by the arrest of toxic phenomena and favorable changes of the character of the exudate when "the lung which was strongly collapsed recovers its ability to reexpand." He concludes that the persistence of respiratory movements of the chest without corresponding movements of the lung, facilitates the production of exudate, while the partial compensation of these movements by inspiratory expansion and expiratory retraction of the lung favors the resorption of exudates.

This mechanism seems, however, to be in contradiction to the unfavorable influence upon the evolution of the old exudates ascribed

by the same author, "to the abandonment of these empyemata to themselves." In fact, in the latter cases according to Monaldi "steady regime of positive or negative pleural pressure is created. The functional antagonism of lung-chest wall movements, which favors the production of exudates, is checked. Further precipitation of fibrin is facilitated partly by surface concentration, as in all colloidal solutions that are allowed to stand undisturbed, and partly because the fibrinogen issuing from the vessels, finds with ease the thrombin ferment produced by the destruction of white cells. The deposition of fibrin causes thickening of the parietal and visceral pleurae and decreases the traumatizing effects of the respiratory movements." If this is true, why then does Monaldi advise active washing of these empyemata with the objective to prevent thickening of the visceral pleura and immobilization of the lung? Also why does he consider reexpansion of the lung as a favorable factor? And how can he accept, in contradiction to what he said before, that conditions for development of fluid are less favorable in these cases although the lung is completely immobilized, and there is no compensation whatsoever to the thoracic movements which, though decreased because of the thickened parietal pleura, still present a considerable extension. Regarding the absorption of purulent exudate, Monaldi formulates a number of interesting, but unwarranted, remarks. Absorption, he says, can take place either by osmosis and diffusion of water and water-soluble salts, polypeptides and amino acids, or by increased permeability of the pleura to proteins. The latter, however, is far from being proved although, he adds, "toxic phenomena can be explained by the absorption of proteins in colloidal state causing foreign protein reaction. Conversely, thickening of the pleurae by deposition of fibrin may totally suppress absorption of exudate. Therefore," he says, "mechanical removal of fibrin may restore the absorbing power of the serosas." The following experiment is quoted by Monaldi in favor of his conception: "When a solution of iodoform is injected into an old empyema cavity, iodoform would appear in the urine after six or eight hours. Following repeated washings of the same cavity, iodoform would appear in the urine after only two to four hours. Therefore," concludes Monaldi, "cures of empyema can occur when the substances present in the exudate are transformed from the colloidal to the water-soluble state, and when the pleural serosa has conserved, or it has regained its normal or near normal permeability."

In closing the chapter of biology of tuberculous empyemata, Monaldi gives the most involved description of "regressive and reparative phenomena." He insists that the intensity of the exudative processes and the limitation and insufficiency of the reparative phenomena in tuberculous empyemata are different from those in acute nontubercu-

lous infections of the pleura. "If tuberculous purulent exudate," he says, "is not removed, but is allowed to stay, it causes marked changes in the circulation and in the histiocytic elements. Thus, the development of curative processes is inhibited. On the contrary, removal of the exudate and washing out of the products of alterations of albumins prevent physical and chemical changes of the pleura and favor the development of neoformative processes which bring about the obliteration of the pleural space." Monaldi even goes further and states that the exudate, "if not removed, may cause secondary fistulae either pleuropulmonary or even thoracic." Consequently, according to this author, systematic washing of the pleura is the treatment of choice in tuberculous empyemata.

The ideas of Monaldi are supported by several European authors. Extensive literature on this subject can be found in Pierre Weiler's[16] monograph on "Lavages of the Pleura." We shall discuss these opinions later.

Another important point in the study of tuberculous exudates is their frequency in pneumothorax as compared with their great rarity, mentioned above, in tuberculous cases without pneumothorax. J. I. Weisman[17] (1936) has found effusions in 84 per cent of 150 consecutive cases of pneumothorax examined by him. Of these, 48 per cent had considerable amount of fluid, and 67 per cent of these patients had far advanced tuberculosis. In 75 per cent, effusion appeared within the first six months and 91 per cent within the first year. In the statistics of Dumarest and Brette,[18] effusion developed in 86 per cent, in Hayes,[19] 82 per cent, in Nablant's,[20] 50 per cent clinically and 100 per cent by x-rays, etc. These figures present an impressive constancy in the statistics of the different authors. It is of interest to mention the observation of Weisman[17] on the "Sequence of Symptoms" preceding the development of fluid. "The onset," says Weisman, "may closely simulate that of ordinary grippe with general malaise, sharp rise in temperature, anorexia, nausea and vomiting; when a large amount of fluid has developed, dyspnea, a feeling of tightness, and palpitations are present. Fluoroscopy may reveal fluid at this time, or it may develop after two or three days. Fluid also should be suspected, if during the refills *it takes a much smaller volume of air than in previous refills to attain the usual intrapleural pressures.* In our series," says Weisman,[17] "79 per cent had a symptomatic onset. However," he adds, "all of these cases had pneumothorax under negative pressures. Most of them had a sudden and unexplained rise in temperature, from 100° to 102°, with an average duration of two weeks. Of 23 patients whose fluid was examined, ten had purulent fluid, and seven of the remaining thirteen who originally had clear fluid became purulent."

In discussing the pathogenesis, Weisman[17] points out the importance of adhesions, and states that in all of his cases in which fluid developed, adhesions were present. He believes that when tubercles are present in the base of implantation at the lung of these adhesions, their rupture under the action of pneumothorax may be responsible for the infection of the pleura. In discussing theories advanced by other authors, such as influence of low temperature of the injected air, or the mechanical irritative effect of air upon the pleura, or of small particles of tissue occasionally introduced with the needle, he dismisses them and believes that they play no part in the production of the effusion.

Joannides[21] believes that effusion may result from an exogenous infection carried into the pleura by the needle, or endogenous, the most important being rupture of the adhesions. Furthermore, he has shown, by his excellent photomicrographic studies of the surface of the lung, the frequent presence of tubercles on the surface of the lung, which he says "*may break off and infect the pleura by the same mechanism as rupture of an abdominal viscus produces peritonitis.*"

Pinner, Moerke and Saley,[22] on the contrary, believe that persistence of pleural effusions is not due to any chemical changes but to changes in the permeability of the pleura which renders impossible the absorption of the tuberculous exudate.

II. MORBIDITY AND MORTALITY

A study of the statistics shows that about 18 per cent of all effusions become purulent. To quote only a few: Hayes[19] reports 21 per cent; Naveau[23] 20 per cent; Dumarest and Brette[18] 16 per cent; Burrell[24] 11.6 per cent; Matson and Bisaillon[25] 12.2 per cent, etc. These figures which may appear surprisingly high to any phthisiologist with large private practice, must be correct, and all authors agree upon them. These figures show that the morbidity of pneumothorax is much higher than it is generally believed, and that the clinical relation of clear fluids to mixed infection empyema is much closer than many a phthisiologist believes. It is natural that we, thoracic surgeons, are becoming increasingly concerned with the constantly increasing number of empyemata in even moderately advanced cases, which become hopeless because of the subsequent development of mixed infection empyema.

Although different authors are using different and various classifications of tuberculous empyemata, they all admit that clinically there are only three well distinct forms: (1) Pure tuberculous empyema with negative sputum; (2) Pure tuberculous empyema with positive sputum, and (3) Tuberculous empyemata with pyogenic superinfection. The mortality depends upon the pulmonary lesions and especially upon the presence or not of mixed infection and upon the treatment used.

With the so-called conservative methods of treatment of these empyemata, namely aspiration, alone or with air replacement, or with irrigation either with saline or other antiseptic solutions, the mortality is around 40 per cent for pure tuberculous empyemata with positive sputum and advanced disease, and around 80 per cent or more for the mixed infection ones. On the contrary, in pure tuberculous empyemata without active pulmonary tuberculosis and without mixed infection, the mortality when adequately treated is hardly 7 to 10 per cent and the cures around 70 per cent (Jones and Alexander[26] report 78.5 per cent).

On the other hand, in all cases of tuberculous empyemata, pure or infected, the best results have been obtained with the radical collapse methods (thoracoplasty and Schede operations). The statistics of Hedblom,[27] of Jones and Alexander,[26] of the Committee of Tuberculous Empyema of the American Sanatorium Association,[28] of Berry,[29] etc., showed that with thoracoplasty the mortality fell to around 25-30 per cent, whereas cures rose to 70-75 per cent.

These figures show very clearly that there is a great discrepancy between the mortality of pure and infected tuberculous empyemata, between empyemata with healed (negative sputum) and active (positive sputum) pulmonary lesions, between empyemata treated with conservative methods, and empyemata treated with thoracoplasty or Schede operations. These facts, which are well known and generally agreed upon, can be neither accidental nor without significance. One may go further and suspect, with reason, that a thorough investigation of these discrepancies may lead to the one still unknown real etiologic factor which determines the production of these empyemata and regulates their evolution.

Certainly we cannot accept as satisfactory solutions to this problem such vague statements as "traumatizing effects of respiration," "changes in the pleural pressures," "irritating effects of the air," "loss of immunologic balance," or "changes in the constitutional resistance of the patient." It is, therefore, noteworthy to submit these theories to strict scrutiny and to endeavor to solve the problem by rigid scientific methods supplied with strictly proved data and tangible evidence.

DISCUSSION

It is evident that all the etiologic factors of the production of empyemata so far proposed cannot be of equal importance, since some of them are found in a number of cases but are lacking in others. Such is the so-called "traumatizing effect of respiratory oscillations upon the pleura," advanced by Monaldi.[13] We cannot accept that this factor is of any great importance in the production of effusion and empyema when we see that in a number of pneumothoraces in which 80 per cent collapse has been obtained for months or years, no

effusion to speak of has developed. Likewise "changes in permeability of the pleura" cannot be the cause of empyemata, since these changes are obviously the effects of empyema and never precede them. On the other hand, the theory that "vascular and circulatory changes can be the causative agent of empyema in pneumothorax," was never corroborated by clinical or experimental evidence. Specific vascular lesions in tuberculosis lead to obstruction and not to vascular dilatation. Vascular failure due to cardiac deficiency should produce bilateral, and not unilateral, pleural fluid. Moreover the heart, in the greatest majority of cases of pneumothorax with fluid, shows no myocardial or functional decompensation. Last but not least, collapse of the lung does not cause hyperemia or venous stasis, but on the contrary, ischemia of the lung due to collapse of its capillaries. If the theory of hyperemia and stasis were true, pleural fluid should develop in all cases of spontaneous nontuberculous pneumothorax, or, at least, in all cases of tuberculous pneumothorax, since in all pneumothoraces these mechanical cardiovascular changes should be present. Likewise, how would this theory explain that effusions are not proportionate to the degree of collapse by pneumothorax but, on the contrary, that they are more frequent in incomplete and limited pneumothoraces?

The same objections are applicable to the theories of "sudden changes of intrapleural pressures being the cause of effusions." It will be shown later what is the real cause of this phenomenon.

The dismissal of these theories clears the ground and limits the discussion to only two etiologic factors: Mechanical and inflammatory irritation of the pleural serosa.

It does not seem that mechanical irritation can be the real cause, because it is far from causing exudate with any degree of constancy. Not only does many a pneumothorax remain for a long period without any fluid or with very little of it, but even fluid is often absorbed although refills are continued. After intrapleural pneumonolysis, when this operation is correctly performed and injury to the lung is avoided, at least in 40 per cent of all cases very little or no fluid develops although the pleura was exposed to high temperatures, to cutting, and to rather rough handling. Likewise, spontaneous nontuberculous or diagnostic pneumothoraces seldom cause pleural fluid. Thus, mechanical irritation of the pleura by the air of pneumothorax cannot be the answer to our problem.

Let us now consider inflammatory irritation of the pleura. All authors admit that production of pleural exudate is intimately connected with the condition of the lung, and that the incidence of pleural exudate is proportionate to the degree and location of the pulmonary lesion, be it tuberculous or of any other nature.

Our problem then becomes limited to the investigation of the transmission of this infection to the pleura. We do not think we should consider here the few and problematic cases of effusions, with tuberculosis limited to the pleura, in which infection was transmitted to the pleura through the lymphatics or the blood vessels. It has been proved, on the other hand, that all cases of the so-called "idiopathic pleurisies" are of tuberculous nature, and that small pulmonary lesions are present. The excellent experimental work of Lemon[30] has shown the close connection between pleural and pulmonary tuberculous infection.

However, although infection of the pleura is, as a rule, accompanied by the development of fluid, the character, amount, and nature of which vary with and depend upon the nature, dosage and virulence of the infecting agent, we are not justified in concluding that all pleural fluids in pneumothorax are due to infection, tuberculous or otherwise, unless we are able to prove the constant presence of this infection and the mechanism by which it is produced.

In a number of cases the mechanism of this infection is obvious. Such are the cases of exogenous pyogenic infection, or when spontaneous pneumothorax develops, and the presence of bronchial fistula can be corroborated by the expectoration of dye injected into the pleural cavity. It was shown above that this mode of infection of the pleura was well known and well described even by the old phthisiologists. Nobody denies this mechanism of pleural infection.

The discussion begins with cases in which no definite clinical signs of spontaneous pneumothorax were noticed, and especially when the presence of bronchial fistulae could not be proved by the clinical or laboratory methods at our disposal.

Although careful scrutiny of the histories of empyemata discloses that, in almost all of these cases elevation of intrapleural pressures and elevations of temperature were noticed just preceding the changes in the appearance of existing serous fluid or the development of purulent exudate, no retrospective diagnosis of bronchopleural fistula can be made as long as the dye injected into the pleural cavity was not expectorated with the sputum. It is precisely in these cases that gas analysis of the pleural air has proved to be of inestimable value. This procedure, by its accuracy and sensitiveness, has permitted us to disclose the presence of bronchial fistula, and to study the phenomena occurring in the pleura which lead to the development of purulent fluid. Serial gas analyses have permitted us to detect the moment of the production of bronchial fistula, often at a time when clinical symptoms or signs were still absent. Thus we believe that we possess today a method which can detect with certainty the presence of pleuropulmonary communication at its earliest stage, and the lack of which was deplored by Monaldi,[13] as said above. Moreover, we believe that this method renders it possible to foresee, with a degree of accuracy

unknown so far, the subsequent course of the empyema, and thus to base the therapeutic indications upon solid foundations. The findings and data obtained by this procedure have led us to our present conception of the pathogenesis of tuberculous empyemata.

III. THE CONCEPTION OF THE AUTHOR

It is our contention that in pulmonary tuberculosis, the production of purulent exudate in the pleural cavity, or the change of existing clear effusions to purulent ones, is *always due to the development of pleuropulmonary fistulae.* These fistulae are caused by the ulceration of caseated subpleural pulmonary tuberculous foci, and lead to the creation of pleuropulmonary communications. It does not seem of any particular interest whether we should lay the emphasis upon the ulcerocaseating pleuropulmonary lesion, or upon the resulting bronchial fistula, since the latter cannot develop without the former and *vice versa.* The mechanism and the pathology of these ulcerations have been well studied by the old phthisiologists as was already mentioned, and they are universally accepted. It is obvious that the breaking up of a caseated tuberculous focus into the pleural cavity is bound to produce tuberculous infection of this cavity.

In the absence of pneumothorax, subpleural tuberculous lesions most often lead to the formation of adhesions which prevent the development of spontaneous pneumothorax and empyema. On the contrary, when the pleural cavity has been transformed from a virtual to a real one by artificial pneumothorax, it is obvious that there is no obstacle to the formation of spontaneous pneumothorax and large size empyema.

In the history of these fistulae, there is a point which is, in our opinion, of the highest importance and which has never been emphasized. This is the significance of the size of these fistulae. Clinical symptoms, surgical signs, laboratory and postmortem data have taught us to distinguish three different sizes: punctiform (less than 0.5 mm.), small (less than 1 mm.), and large (1 to 10 mm. or more). This division was based upon the data obtained by the gas analysis procedure, as will be shown with the description of the technique of the latter.

The study of the pathologic physiology of these fistulae is of paramount importance. It throws, we believe, a new light upon the whole question and opens new vistas on the pathogenesis, prognosis, and management of tuberculous empyemata.

1. *Punctiform Fistulae.*—The small size of these fistulae is due to the smallness of the caseated tuberculous focus. These fistulae represent shallow erosions of the pulmonary parenchyma. Therefore, only superficial alveoli and small respiratory bronchioli open into the pleural cavity. It is obvious that because of the small size of the initial caseous lesion, the dosage of the contamination and the degree

of the infection of the pleura are slight. On the other hand, the small size of the perforation causes only slight modification of pleural pressures. Functional respiratory symptoms are so slight that they may not be noticed, especially when we are not on the lookout for them. However, the diameter of these punctiform fistulae changes during the respiratory cycle. It is reduced during expiratory retraction of the lung and increased during inspiratory expansion. Thus is becomes easy to understand that air can be sucked into the pleural cavity during inspiration, whereas pleural fluid and dye, injected into the pleura for diagnostic purposes, cannot pass into the bronchial tree. This is further proved by the fact that in these cases when the dye is injected into the bronchial tree instead of the pleural cavity, it often passes into the pleural cavity.

However small this fistula may be, it can always be detected by the gas analysis procedure. In fact, previous work has shown[5] that the respective amounts of O_2 and CO_2 in the pleural air are constant in each of the three possible conditions of pneumothorax, namely, without fluid, with straw-colored fluid, or with purulent exudate. In the absence of fluid, the O_2 is about 3 to 4 per cent and the CO_2 about 6 per cent. When straw-colored fluid is present, O_2 drops to between 1 and 2 per cent and CO_2 rises to between 6 and 8 per cent. When purulent fluid is present, O_2 is less than 1 per cent and CO_2 above 8 per cent. These percentages show a remarkable steadiness.

It is obvious that in each of these three conditions O_2 and CO_2 percentages can vary only toward one direction, namely, the increase of O_2 and the decrease of CO_2; and this can be due only to one cause: bronchial fistula. The composition of the alveolar air is O_2, 15 per cent, and CO_2, 6.0 per cent; and of the atmospheric air O_2, 20.7 per cent, and CO_2, 0.03 per cent. Whether a small bronchial fistula is formed when the pleural cavity will communicate with alveolar spaces only, or a large bronchial fistula is present when the pleural space will receive air from larger bronchi which contain atmospheric air, it is evident that in both cases the O_2 of the pneumothorax air will increase while its CO_2 will decrease. Since these changes are proportionate to the size of the fistulae, it is obvious that the size of the fistulae can be deducted from the corresponding increase of O_2 and decrease of CO_2.

Small fistulae give rise to slight tuberculous infection of previous aseptic straw-colored fluid (below Gaffky 2). They cause small rises of intrapleural pressures so that clinical signs of spontaneous pneumothorax are, as a rule, absent. Changes of chemical composition of air, although always present, show slight deviations. Only alveoli and small bronchioli, generally aseptic, open into the pleura. Therefore, pyogenic infection is exceptional or, if present, very slight. Lastly, the small size of the fistulae and the exiguity of the causative caseous focus, render spontaneous obliteration of these fistulae easy. Thus most

often the course of pneumothorax, temporarily disturbed by the development of small bronchial fistula, returns to normalcy and resumes its previous course. Following closure of the fistula and decrease of O_2, tubercle bacilli may disappear from the pleural fluid; the latter can, even when it had become slightly turbid, change again to clear straw-colored fluid and gradually become absorbed. Conversely, the same "acute episode" may be repeated and followed either by the same sequence of effects, or, if the fistula becomes larger, give rise to symptoms described below. We think that this conception presents a rational explanation of the "slight and transient complications" with minimal clinical symptoms which often develop in the course of pneumothorax. In fact, changes of the composition of the pleural air can show in all these cases the presence of bronchial fistula. If this conception be true, there is neither necessity nor room for metaphysical and involved theories. The etiology becomes clear, easy to grasp, and easy to prove, and the clinical and pathologic evolution of the disease proceeds according to simple and fundamental physical, chemical and biological laws.

2. *Fistulae of Moderate Size.*—In these cases clinical symptoms and laboratory findings are different. We deal here with larger subpleural caseated foci, and larger and deeper fistulae. The dosage, therefore, of tuberculous infection of the pleural cavity is greater than in the previous form. The fluid becomes turbid or frankly purulent and richer in tubercle bacilli. The amount of air passing into the pleural cavity is larger; therefore, there is more marked rise of O_2 and a greater drop of CO_2. Rises of intrapleural pressures are more marked, and clinical symptoms of spontaneous pneumothorax are more apparent. Furthermore, the pleural purulent exudate can pass into the bronchial tree more easily than in punctiform fistulae. Thus the amount of sputum is increased and is more uniformly purulent. Dye injected into the pleural cavity, as a rule, passes into the sputum. But the most important complication in these cases is that larger bronchi are opened into the pleural cavity, and thus pyogenic superinfection of the tuberculous fluid is more frequent. However, even these fistulae may occasionally close, although this eventually is rarer than in the first group; the toxic symptoms may disappear and even the fluid may be reabsorbed. Naturally the chances for spontaneous closure decrease as the size and depth of these fistulae increase.

3. *Large Fistulae.*—This variety develops as a rule in far advanced cases of pulmonary tuberculosis, badly controlled by incomplete pneumothoraces. Gas analysis shows that the pleural air takes almost the composition of atmospheric air. The large size of the causative caseated pulmonary foci produces massive tuberculous infections and the great amounts of oxygen present in the pleura favor growth of tubercle bacilli in the fluid. Considerable amounts of pleural exudate pour

into the bronchial tree and cause rapid and extensive spreads in the healthy parts of the lungs. When we add to these disturbances the progressive anoxemia of the patient, the excessive toxicity due to severe mixed infections, and the rapidly increasing amyloidosis, we can easily understand the dramatic loss of weight and the swift decline and exodus of patients who, a short while before, were still in fair condition.

EVIDENCE IN FAVOR OF THE CONCEPTION OF THE AUTHOR

The evidence that we are about to present in favor of this conception is clinical, surgical, pathologic, and laboratory.

1. *Clinical Evidence.*—We shall consider here the evidence in punctiform and moderate size fistulae alone. The large fistulae need no discussion.

The above symptoms described as the "acute episode" preceding the change of a clear effusion into turbid or purulent fluid can always be found when we look for them. This "acute episode" is characterized by chest pain, elevation of temperature, increase of intrapleural pressures, and modification of the composition of the pleural air. These changes may be very slight and temporary, but they are always present. In going back over the histories of cases with empyema, we were always able to find these symptoms present at the time that clear fluid began to turn to purulent. In the past we had not noticed them simply because we did not look for them. This explains the impression of Morelli[14] that infection of the pleura may take place through normal and intact visceral pleura; but also it justifies the hypothesis of Monaldi,[13] mentioned above, that "bronchial fistulae are perhaps more frequent than we believe, because, unfortunately, we do not possess any method by which we may discover them." Gas analysis, by giving us this missing procedure, not only has shown the frequency of these fistulae but, even more, it has proved their constant presence.

Other clinical evidence is that in punctiform fistulae, which can easily become obliterated, these "acute episodes," contrary to larger fistulae, are most often only transient and of short duration.

In favor of this conception is also the fact that the largest number of these complications arise during the first three or four months of pneumothorax and especially when the collapse is not efficient. The explanation of this phenomenon is, we believe, that when pneumothorax has produced successful selective collapse of the diseased lung the improvement of the disease is rapid and ulcerations of the subpleural foci are less liable to occur. On the contrary, when collapse is incomplete, pulmonary lesions progress, and subpleural foci are more exposed to caseous ulceration.

The relatively frequent development of purulent fluid when positive pressures are used in order to "force" or "tear" pleuropulmonary adhe-

sions, offers another evidence in favor of our conception. In fact, pleuropulmonary adhesions between parietal and visceral pleurae are formed before collapse of the lung in places in which superficial pulmonary lesions come in contact with the parietal pleura. Therefore, a peripheral pulmonary lesion is necessarily always present at the pulmonary part of each adhesion. When artificial pneumothorax is induced, it tends to separate the visceral from the parietal pleura. The former, being intimately attached to the pulmonary parenchyma, cannot yield. On the contrary, the parietal pleura, being only loosely attached to the endothoracic fascia, is easily pulled out by the retracting lung. Thus thin or thick folds of the parietal pleura are formed, representing the so-called pleuropulmonary adhesions. It is obvious, therefore, that the weakest point of each adhesion is at its pulmonary attachment and corresponds to the site of the initial pulmonary lesion. Thus when an adhesion breaks it will be at that point, and this renders possible the production of a bronchial fistula.

Other evidence in favor of this conception is that often marked dyspnea and elevation of intrapleural pressures follow rapid aspiration of pleural fluid. It is very probable that in these cases a small fistula situated below the level of the fluid may have remained closed because of the pressure exerted upon it by the column of the fluid above it. To be more precise, air cannot pass from the bronchi into the pleural cavity unless the negative pressure in the latter is superior to the pressure exerted by the fluid column above the level of the fistula. Ornstein[31] has shown that dyspnea and cyanosis may be rapidly relieved in these cases when the chest is filled up again with saline solution.

It is well known that patients with the chest full of pus may do well for months or years. This is true, and its explanation becomes easy with our conception. The fact that the chest is filled up and no air is present shows that there is no open bronchial fistula. In these cases, gas analysis had invariably shown the lowest O_2 (around 0.8 per cent) and the highest CO_2 (around 15 per cent). It is obvious that in these cases there cannot be any pleuropulmonary communication. It is altogether different in the chest "half full of pus." The general condition of these patients is less satisfactory, the fluid is reproduced more rapidly, frequent aspirations are necessary, and the fluid contains large numbers of tubercle bacilli. Invariably in these cases gas analysis had shown constantly, or intermittently, high percentages of O_2, and low percentages of CO_2. It is evident that these changes are due to temporary openings and closures of the fistula. Ornstein[31] believes that in these cases the fistulae may become opened or closed by the fluid according to the position of the patient, and gas analysis has often corroborated this hypothesis. The disappointing results obtained by washing the pleural cavity with antiseptic or bacteriostatic sub-

stances, with the hope of rendering it aseptic, can now be easily explained. The innumerable substances used for this purpose prove the inefficiency of the procedure. It is not without interest to mention here a few of the substances which have been used for pleural lavages: normal saline solution, colloids of gold (Villaret and Evans), copper or silver (argyrol, argochrome); formalin or iodoform in glycerin (Murphy, Kohn[32]); lipiodol, ethyl morrhuate, allochrysin (Piechaud[33]), methylene blue (Rosenstein,[34] Stivelman), gentian violet (Singer, Wells), Lugol's solution (Piéchaud,[33] Weiler[16]), myrol, solganol B; insufflations of vapor of azote-gomenol (Ballon), potassium permanganate (Ulrici[35]), acriflavine (Hayes[19]), trypaflavine (Michels[36]), Dakin's solution (Burrell,[24] Dumarest,[37] Guinard[38] and Jullian), vuzin (Leschke[39]), rivanol (Bachmeister[40]), simple oil or containing gomenol, vaccines and bacteriophages.

Careful study of the numerous papers in which the curative qualities of the substances mentioned above were reported, shows that the good results vary between 15 and 25 per cent and that these percentages are about the same whatever the respective substances used. The fact that simple washing with plain saline solution yielded about the same results as bacteriostatic dye (gentian violet, methylene blue), colloidal solutions or strong antiseptics (Dakin's solution, azochloramide and even iodine or formalin), leads to the conclusion that the good results reported cannot be ascribed to the chemical properties of these substances. On the other hand, the symptoms enumerated by these authors as preceding the improvement of these patients point to the real factor of cure. These symptoms are decrease of intrapleural pressure, tendency of the lung to expand, decrease of the numbers of tubercle bacilli in the pleural fluid, histologic changes of the morphologic elements of the fluid and decreased expectoration. There can be no doubt that the symptoms indicate obliteration of the pleuropulmonary fistula.

Conversely, the symptoms given by the same authors as announcing recurrences of the disease after a period of quiescence, again point to the reopening of the bronchial fistula. These symptoms are rise of intrapleural pressures, dyspnea, increase in number of tubercle bacilli in the fluid, fever, and toxic phenomena. A number of authors mention particularly that methylene blue which has been previously injected into the pleural cavity, and which did not show in the sputum during the period of improvement and quiescence, appears in the sputum with the recurrence of symptoms. This is a decisive evidence of the reopening of the fistula.

Regarding changes in pleural pressures always present at the time of the "acute episode," which mark the transformation of a "cold" and quiescent effusion, according to the French terminology, to a "hot" empyema, enough was said already. In cases with punctiform

fistulae, it was only noted that less air was used in order to bring the pressures to the usual height, while in larger size fistulae spontaneous pneumothorax often with high positive pressures was reported. It is peculiar that most of the authors had not recognized so far a real relation of cause to effect between bronchopleural communication and empyema, even in cases with obvious spontaneous pneumothorax.

2. *Surgical Evidence.*—In all of our cases of tuberculous mixed infection empyema upon which Schede operations were performed, bronchial fistulae were always present. In a limited number in which toxic symptoms had more or less completely subsided, we have found, invariably, narrowed or even closed bronchial fistulae.

After the parietal pleura was incised and the visceral pleura was exposed, we had covered the latter with saline solutions; bubbling of air during expiration indicated the position of the fistulae. Without this technique it was often impossible to discover the fistulae because of the irregular surface of the thickened visceral pleura.

Last but not least, the surgical evidence includes the striking superiority of the curative results obtained in tuberculous empyemata by thoracoplastic collapse over all other methods which do not produce mechanical collapse and obliteration of the pleural cavity. No other explanation can be given for these good results of thoracoplasty and Schede than the definite closure of the bronchial fistula and the obliteration of the pleural cavity. Conversely, persistence of these fistulae, in cases of insufficient thoracoplastic collapse, as it occurs when the parietal pleura is very thickened and uncollapsible, caused failure of cure of the empyema by thoracoplasty. Only a Schede operation and covering of the visceral pleura with a pedunculated muscular flap could lead in these cases to the obliteration of the fistulae and to definite healing of the empyema.

3. *Pathologic Evidence.*—Cases of empyemata that have come to autopsy and in which during life neither elevated intrapleural pressures were noted nor dye injected into the pleural fluid was expectorated, were most instructive in showing the presence of bronchial fistulae. These fistulae, however, were always either very small or obliterated, and a careful search was necessary to find them. This explained the absence of elevated pleural pressures, of toxic phenomena and expectoration of the dye. Bronchial fistulae were always present in severe empyemata, especially with mixed infection. They present at the autopsy various forms and shapes. They were round, oval, or irregular. In recent cases they opened in the midst of caseated pleuropulmonary foci. In older cases they were limited by fibrotic tissue. In others they presented smooth contours. Healed fistulae could be found in empyemata in which the acute symptoms had subsided for a long time.

We are indebted to Dr. Auerbach, director of the Pathological Department of Sea View Hospital, for this information.

Another evidence in favor of our conception is offered by the striking likeness of the pathologic physiology of tuberculous cavity of the lung with the cavity of tuberculous empyema. They both represent tuberculous abscesses with the only difference that the former were developed in the lung parenchyma whereas the latter were developed in the pleural cavity. The mechanics and biology of both are identical. As long as a pulmonary cavity is connected with the bronchial tree

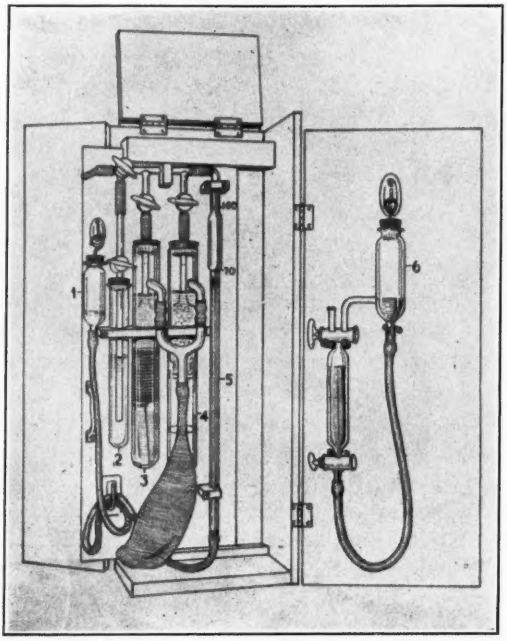


Fig. 1.—Coryllos' clinical apparatus for gas analysis of the pleural air.

Air is aspirated from the pleura into a Haldane sampling tube (6), the capacity of which is about 20 c.c.—10 cm. of this air is introduced into the graduated buret of the apparatus (5). This air is passed successively into tube 4 containing a solution of sodium hydroxide in which carbon dioxide is absorbed. Then the same air is passed into tube 3 containing metallic copper in a solution of ammonium chloride in ammoniac. This solution absorbs the oxygen of the air. Thus by two successive readings in the buret, it is easy to determine the amount of oxygen and of carbon dioxide contained in the pleural air.

by open bronchial outlets, it cannot close and the tubercle bacilli grow in it actively because of adequate oxygen supply; the sputum passes into the bronchial tree, contaminates the lung, and causes bronchiogenous spreads of the disease. Conversely, closure of the bronchial outlets of a pulmonary cavity produces obliteration of the cavity and clinical cure by the absorption of the air contained in it, oxygen starvation of the tubercle bacilli, and discontinuance of the passage

of the latter into the bronchial tree. The same mechanic and biologic changes are observed in the empyema cavity. When pleuropulmonary fistulae are present, the pleural cavity cannot become obliterated because the lung cannot reexpand; tubercle bacilli grow freely in the pleural fluid because of adequate supply of oxygen; highly tuberlized fluid passes into the bronchial tree favoring bronchiogenous spread of the disease. Closure of these fistulae stops the entrance of air into the pleural cavity, thus favoring expansion of the lung and obliteration of the pleural cavity; on the other hand, the danger of bronchiogenous spread disappears because pleural pus cannot enter into the bronchial tree. Furthermore, the number of tubercle bacilli in the fluid decreases rapidly as the oxygen supply vanishes.

This peculiar likeness between tuberculous cavities and empyema cavities, never stressed before as far as we know, represents an important evidence in favor of our conception.

4. *Laboratory Evidence.*—We have already insisted upon the increase of the number of tubercle bacilli in the pleural fluid, the histologic and chemical changes of the latter, and the appearance of pyogenic organisms. It is evident that the only possible explanation of these changes in the absence of thoracic fistula is offered by the development of a pleuropulmonary fistula.

We shall insist more particularly on the evidence offered by systematic and serial gas analysis of the pleural air, and give a brief description of the technique of this procedure.

This procedure can be carried out with any of the gas analysis apparatus used in the laboratory. However, in order to simplify the technique, we have designed a portable apparatus which renders possible gas analysis with results accurate enough for clinical purposes at the bedside of the patient* (Fig. 1).

Technique.—Ten cubic centimeters of air are aspirated from the pleural cavity and introduced into the apparatus. Aspiration can be performed either by means of a Haldane mercury sampling tube or simply by a 10 c.c. syringe well lubricated with paraffin oil. This air is introduced into the apparatus and passed successively through a solution of sodium hydroxide where CO_2 is absorbed, and then into a solution of pyrogallic acid, or of ammonia-ammonium chloride in which metallic copper is placed. Here O_2 is absorbed. By measurements in the graduated buret of the instrument of the volumes of gas after each operation, it is easy to calculate the respective percentages of O_2 and CO_2 with an approximation to a second decimal. The operation is simple, easy to master, and can be performed in ten minutes.

As an example, we shall give here the figures of one of our patients before and after fluid had become purulent.†

*This apparatus is manufactured by Elmer and Amend, New York, N. Y.

†Figures communicated by Dr. Welkind and Dr. Matzusawa working on this question in the Division of Tuberculosis of Metropolitan Hospital, N. Y. (Director G. G. Ornstein.)

TABLE I

DATE	O_2	CO_2	METHYLENE BLUE	BACTERIOLOGY TUBERCLE BACILLI	PYOGENES	PLEURAL PRESSURES
3/31/36	ONSET OF CLEAR FLUID					
5/14/36	1.5	9.0	-	G.2	0	
9/13/36	1.0	10.5	-	G.2	0	-14-6
8/24/36	14.2	3.8	+	G.3	+	+2+3
10/12/36	17.5	1.5	+	G.3	+	+2+3
10/27/36	17.3	2.7	+	G.8	+	+2+3
11/18/36	19.6	1.78	+	G.8	+	+2+3

This patient, a white male, thirty-four years of age, had advanced tuberculous lesions with multiple cavities on the left side. Pneumothorax produced an incomplete collapse. Positive sputum became negative in March, 1936, and remained such until September, 1936. During this period the temperature was normal and no toxic symptoms were present. A small amount of straw-colored fluid was present. Oxygen was 1 to 1.5 per cent only, whereas CO_2 was 9 to 10.5 per cent. Pleural effusions presented a low grade of tubercle bacilli (Gaffky 2). Pleural pressures were strongly negative (1-14-6).

Suddenly the patient complained of a chest pain, dyspnea, and chills. The pleural pressure became positive (-2-3), and the temperature rose to 102.5°. The pleural fluid rapidly became purulent while the sputum increased in amount. In addition, the sputum assumed a purulent appearance similar to the appearance of the pleural exudate, and turned positive again for tubercle bacilli. At the same time the O_2 rose to 14.2 per cent, while CO_2 dropped to 3.8 per cent. The intrapleural pressures remained positive, the percentages of O_2 high, and of CO_2 low. The sputum remained positive, and the Gaffky of the sputum was the same as of the pleural exudate which rose to Gaffky 8. The temperature presented septic spikes, and the general condition declined very rapidly. Pyogenic infection followed immediately after the appearance of these symptoms. The high O_2 and low CO_2 indicate a large bronchial fistula which explains the almost atmospheric intrapleural pressure and the rapid development of pyogenic infection. It was easy, therefore, to diagnose a rather large pleuropulmonary communication and predict a severe prognosis. This patient died.

PRACTICAL APPLICATIONS OF THE CONCEPTION OF THE AUTHOR IN THE MANAGEMENT OF TUBERCULOUS EMPYEMATA

If our ideas on the pathogenesis of tuberculous empyemata are correct, it is evident that prognosis and therapeutic indications in tuberculous empyemata can be built upon more solid foundations than up to the present time.

1. *Prognosis.*—An accurate determination of the size of the fistula renders possible an equally accurate prognosis of the future course of the disease. When a punctiform fistula is diagnosed and signs of mixed infection are absent, the prognosis is rather good, because of the ease with which these fistulae close spontaneously; therefore, we can wait and carefully follow by gas analysis the course of the fistula. Radical measures in these cases are not urgent because the fistula may close, the fluid may be absorbed and the lung reexpanded. Therefore, the

prognosis and management depend much more on the pleural fistulae than on the pleural fluid itself.

In the presence of moderate size fistulae, the prognosis is worse. In these cases it is of no great importance whether mixed infection is already present or not, and whether the pulmonary lesions are active or not. The important factors in these cases are that spontaneous closure of these fistulae is very unlikely, and that mixed infection is highly possible. Therefore, in these cases thoracoplastic collapse of the pleural cavity should be done without any delay. In fact, pure tuberculous empyemata do respond to thoracoplasty with 70 per cent clinical cures, whereas hardly 20 per cent of mixed infection empyemata can be cured whatever may be the method of treatment. In other words, the treatment of mixed infection empyema should be "preventive." This objective can be reached only by complete and early thoracoplastic collapse.

Thus far the argument was advanced by the phthisiologists, especially in cases of empyema with healed pulmonary lesions and negative sputum, that conservative treatment was a more reasonable procedure. It is true that a number of pure tuberculous empyemata may never give rise to any alarming symptoms and may even become absorbed. This argument, which is perfectly correct in cases with closed bronchial fistulae, has certainly caused the death of a great number of patients with open or reopened fistulae. The impossibility of distinction between the two varieties has been so far the cause of our inability to establish a correct differential diagnosis and consequently formulate the correct therapeutic indications. Today, gas analysis permits us to predict in which empyemata conservative treatment is indicated and in which immediate thoracoplasty may be a life-saving procedure.

2. *Therapeutic Indications.*—If we are right in our conception, therapeutic indications in tuberculous empyema must become as simple and clear as are the therapeutic indications today in pulmonary, bone, or kidney tuberculosis.

We believe that the simplest and most rational clinical classification of tuberculous empyemata is their division into three varieties. No more complicated classifications are necessary. These varieties are (1) Tuberculous empyema with negative sputum. (2) Tuberculous empyema with positive sputum. (3) Tuberculous empyema with mixed infection.

A. *Third Group:* There is little discussion, if any, on the therapeutic indications on the third variety. Whatever may be the kind of infection, aerobic or anaerobic, immediate drainage should be performed, and aided by mechanical and chemical disinfection of the cavity in order to produce a rapid disintoxication and to put the patient in condition to stand thoracoplastic collapse. Continuous lavages with

azochloramide solutions have given us the best results. The earlier this procedure is used, the better it is. However, it is not always necessary that complete disintoxication of the patient be obtained before thoracoplasty has started. This much desired complete disintoxication may never occur and precious time is lost in waiting for it, during which the condition of the patient becomes progressively and rapidly worse. On the contrary, in beginning thoracoplasty early, in proceeding with multiple stages, and by adjusting the extent of each stage to the condition of the patient, we can do more for these patients than we dared expect at the beginning of this investigation, hardly three years ago. Often, toxic patients, who were running high temperatures before the first stage was performed, showed marked improvement immediately following this stage. Temperature and toxic phenomena gradually decreased as the capacity of the empyema cavity was reduced. Thus, after each stage the patients were in a better condition for the subsequent stage. We firmly believe today that this point is of extreme importance in the management of these empyemata, contrary to the routine expectant treatment in which, as a rule, the longer we wait the worse the condition of the patient grows. Moreover, early operation prevents extreme thickening of the parietal pleura, which precludes any successful obliteration of the empyema by thoracoplasty alone and renders more serious operative procedures necessary. By operating early we certainly take chances and expose the debilitated patient to danger; but on the other hand, by this method we have increased our cures to 50 per cent instead of only 20 per cent, and decreased our mortality to 25 per cent instead of 80 per cent.

B. *Second Group:* In cases of empyemata with positive sputum there is little discussion. The consensus of opinion is in favor of immediate thoracoplastic collapse, with the double aim of collapsing the diseased lung and obliterating the pleural cavity. The only point of discussion in these cases is whether drainage should be used before operation or not. Of course by draining we expose the patient to the danger of mixed infection. But on the other hand, especially when the fluid is rapidly reformed, we are faced with a double danger. First, the passage of a considerable amount of pus into the bronchial tree during the operation and bronchiogenous spread, or even asphyxia by drowned lung. This danger is always present, because even the most careful aspiration by the needle cannot insure, in all cases, a complete emptying of the pleural cavity. The second danger is that the rapidly reformed fluid does not allow a complete obliteration of the pleural cavity. As a rule, repeated taps have proved inefficient to keep the pleural cavity empty until the resected ribs were regenerated, and furthermore, they expose to the development of thoracic fistulae by tuberculization of the tract of the needle.

Personally, I perform strictly closed water-sealed intercostal drainage with urethral catheter, the same day or the day before the operation. This drainage should always be performed in the anterior surface of the chest, sixth interspace, in order to avoid interference of the sinus with the incision of the thoracoplasty.

C. *First Group:* Therapeutic indications in the first group, which comprises pure tuberculous empyemata with negative sputum, is the object of the most vivid discussions. If we are right in our conception, this important problem can be easily solved. In fact, indications in these cases do not depend upon pulmonary lesions and the clinical condition of the patient alone, but upon the presence or absence of the patency and the size of bronchial fistula. Thus the surgeon and phthisiologist must be able to manage these cases following the data given by gas analysis of the pleural air. According to the information so obtained, they should be able to conduct the treatment with a feeling of security unknown up to this time.

Only time will show whether our conception is correct. Personally, we believe that it is correct, and this belief is based upon the clinical results obtained up to this date, a detailed analysis of which will appear shortly in a special paper. However, much more work and investigation are necessary; only the application of this conception by the profession will decide upon its merits.

CONCLUSIONS

1. A study of the pathologic physiology of postpneumothorax pleural fluids was carried out, based on the determination of changes of the chemical composition of the air of pneumothorax, of modifications of intrapleural pressures, of physical, chemical, and bacteriological changes of the pleural fluids, of surgical and clinical findings, and on postmortem data.

2. This study has convinced us that the most important factor in the pathogenesis of purulent effusions, or the changes of clear effusions to purulent, is the development of pleuropulmonary communications (bronchial fistulae) due to caseation and ulceration of subpleural tuberculous foci.

3. The other numerous causes of the development of purulent pleural exudates advanced so far have been discussed. They were shown to be of secondary significance, if of any at all.

4. Serial gas analysis of the pleural air has allowed the detection of the presence and the appraisal of the size of the fistulae with a marked degree of accuracy. It was shown that the future course of empyemata was closely connected with, and regulated by, the presence and the size of these fistulae.

5. The larger are these fistulae the more massive is the tuberculous infection and the greater the danger of mixed infection. On the other

hand, their chances for spontaneous closure, frequent in small fistulae, decrease as they become larger.

6. Cases with punctiform and transient fistulae may be treated conservatively.

7. Cases with mixed infection should be treated by drainage, lavages, and early thoracoplastic collapse.

8. Cases with pure tuberculous empyema and positive sputums should be operated upon by thoracoplastic collapse.

9. All other cases should be managed according to the presence, or not, and the size of the fistulae. When moderate or large-sized fistulae are present, early thoracoplastic collapse is the only way of preventing mixed infection.

10. The best means for decreasing incidence and the appalling mortality of mixed infection empyemata is their prevention. In this endeavor, the data furnished by the gas analysis procedure represent a precious guide.

REFERENCES

1. Coryllos, P. N.: Physics Applied to Tuberculosis, J. Franklin Inst. **200**: 287, 1935.
2. Coryllos, P. N.: Pathologic Physiology of the Tuberculous Lung, Goldberg's Clinical Tuberculosis, F. A. Davis Co., Philadelphia, 1935.
3. Coryllos, P. N., and Birnbaum, G. L.: Alveolar Gas Exchanges and Atelectasis, Arch. Surg., Part II, **21**: 1214, 1930.
4. Coryllos, P. N.: Studies in Pulmonary Gas Absorption in Bronchial Obstruction; The Behavior and Absorption Times of Oxygen, Carbon Dioxide, Nitrogen, Hydrogen, Helium, etc., in the Lung and the Pleura, Am. J. M. Sc. **183**: 326, 1932.
5. Coryllos, P. N., Konterwitz, H., and Levine, E. R.: The Practical Applications of Gas Analysis in Artificial Pneumothorax, Am. Rev. Tuberc. **26**: 153, 1932.
6. Grancher, J., and Hutinel, V.: Phthisie Pulmonaire, Dictionnaire des Sciences Méd., Paris, Asselin, Houzeau et Masson **24**: 460, 1887.
7. Laënnec: Quoted by Grancher and Hutinel.
8. Herard and Cornil: Quoted by Grancher and Hutinel.
9. Lendst: Quoted by Grancher and Hutinel.
10. Louis: Quoted by Grancher and Hutinel.
11. Weil: Quoted by Grancher and Hutinel.
12. Jaffé, R. H.: The Pathology of Pulmonary Tuberculosis, Goldberg's Clinical Tuberculosis, F. A. Davis Co., Philadelphia, 1935.
13. Monaldi, V.: La Cura dell'Empiema para-pneumotoracico, Lotta contro la tuberc. **6**: 1127, 1935.
14. Morelli, E.: Sulla genes; dell'Empiema. Sintomatologia della perforazione pulmonare, Boll. d. Soc. med.-chir., Pavia, 1921.
15. Morelli, E., and Perin, L.: Malattie dei bronchi e dei polmoni. Malattie della pleura, etc., Trattato Itliano di Medicina Internat., 1931.
16. Weiler, P.: Les Lavages de la Pleure, Masson et Cie., Paris, 1934.
17. Weisman, J. I.: Effusions Following Artificial Pneumothorax, Am. Rev. Tuberc. **33**: 522, 1936.
18. Dumarest, F., and Brette, P.: La Pratique du Pneumothorax pulmonaire, Masson et Cie, Paris, third ed. 1929.
19. Hayes, J. N.: Purulent Effusions in Artificial Pneumothorax, Am. Rev. Tuberc. **16**: 650, 1927.
20. Nablant, J. P.: Treatment of Pleural Effusions Complicating Artificial Pneumothorax, Am. Rev. Tuberc. **25**: 650, 1932.
21. Joannides, N.: Behavior of Pleural Effusions in Hydropneumothorax, Am. Rev. Tuberc. **29**: 329, 1934.

22. Pinner, M., Moerke, G., and Saley, D. H.: Pleural Effusions, Am. Rev. Tuberc. **22**: 127, 1930.
23. Naveau: Quoted by Weisman.[17]
24. Burrell, L. S. T.: Effusion Complicating Pneumothorax, Brit. M. J. **2**: 8, 1926; Lancet **2**: 1209, 1929.
25. Matson, R. W., Matson, R. C., and Bisaillon, M.: Artificial Pneumothorax, Am. Rev. Tuberc. **9**: 294, 1924.
26. Jones, C. J., and Alexander, J.: Results in 70 Consecutive Cases of Tuberculous Empyema, Am. Rev. Tuberc. **29**: 230, 1934.
27. Hedblom, C.: The Diagnosis and Treatment of Tuberculous Empyema, Surg., Gynec. and Obst. **24**: 445, 1922.
Hedblom, C.: Surgical Treatment of Tuberculous Empyema, J. THORACIC SURG. **2**: 115, 1932.
28. Report of the Committee of End-Results in 235 Cases of Tuberculous Empyema, Tr. Nat. A. Tuberc., p. 340, 1932.
29. Berry, F. B.: Tuberculous Pyopneumothorax with Pyogenic Infection, J. THORACIC SURG. **2**: 139, 1932.
30. Lemon, W. S., and Montgomery, L. G.: Pleural and Pulmonary Lesions Resulting From Intrapleural Injection of Mycobacterium of Tuberculosis, J. THORACIC SURG. **3**: 612, 1933-34.
31. Ornstein, G.: Personal communication.
32. Kohn, H.: Zur. Conservativen Behandlung Tuberkulöser Pleuraexsudate, München. med. Wchnschr. **70**: 1343, 1923.
33. Piéchaud, F., and Cras, H.: À Propos de Quelques Cas de Pleurésies Purulentes Tuberculeuses, Gaz. d. hôp. **105**: 704, 1932.
34. Rosenstein, P.: Methylenblauspülung der Pleuraraumes mit tödlickem Ausgang, Deutsche med. Wchnschr. **51**: 908, 1925.
Rosenstein, P.: Pleura-Empyem und Rivanol, Klin. Wchnschr. **4**: 544, 1925.
35. Ulrici, H.: Diagnostik und Therapie der Lungen und Kehlkopftuberkulose, Springer, Berlin, 1924.
Ulrici, H.: Kritishe Sammelreferate über Pneumothorax-Therapie, Deutsche med. Wchnschr. **49**: 1351, 1923.
36. Michels, G.: Zur Pneumothoraxbehandlung, Beitr. z. Klin. d. Tuberk. **46**: 132, 1921.
37. Dumarest, F.: Les Lavages Pleuraux dans les Pleurésies du Pneumothorax Artificiel, Rev. de la tuberc. **12**: 167, 1931.
38. Guinard, U.: Quelques Constatations à propos des Épanchéments du Pneumothorax, Soc. Scient. de la Tuberculose. (Quoted in Weiler.[16])
39. Leschke, Erich: Offene Pleurapunktion und Pleuraspülung, Klin. Wchnschr. **5**: 2429, 1926.
40. Bachmeister: Lungen Tuberkulose, G. Thieme, Leipzig, 1926.
41. Auerbach, O.: Personal communication.
42. Coryllos, P. N.: The Mechanics and Biology of Tuberculous Cavities, Am. Rev. Tuberc. **33**: 639, 1936.

INTRATHORACIC XANTHOMATOUS NEWGROWTHS*

Report of Two Cases
And the Collection of Three Similar Cases in the Literature

Edgar W. Phillips, M.D.†

THE characteristic histology of a xanthomatous neoplasm is the xanthoma cell or "foam cell." These cells have a wide appearance in pathologic conditions. Much confusion exists as to the origin and significance of these cells.

Ewing[1] states that the structure of a xanthoma shows the great abundance of lipoid globules in large swollen polyhedral cells and smaller spindle cells, and that the material is an orange yellow lipochrome, more of it stained by sudan III than by osmic acid. The globules are doubtly refractive and of myelin nature or probably a cholesterin fatty acid ester. In the process of making microscopic sections this ester is dissolved, leaving vacuoles giving the characteristic appearance of the foam cell. Humiston and Piette[2] compare the golden yellow color of a xanthoma to the corpus luteum or the cortex of the adrenal gland, and believe the color depends upon the accumulation of certain peculiar fatty substances, as doubly refractive cholesterol esters, protagon, and neutral fats. Mason and Woolston[3] disagree with the above opinions as to what produces the yellow color. They state that cholesterol is colorless and that the color comes from carotin and xanthophyll.

The cell was first described by Dor in 1892, and since that time several theories have been advanced as to its origin and significance. Virchow (quoted by Ewing) regarded xanthoma tissue as intermediate between connective tissue and fat tissue. Hallopean and Torok (quoted by Ewing) considered foam cells as equivalent to embryonal fat cells. Buxton[4] thought the xanthoma cell was dependent upon tissue disintegration. Lubarsch (quoted by Wessén) classed tumors of xanthomatous nature as a collateral species of lipoma. MacCallum[5] does not mention tumors such as are described in this report and says that the xanthoma or the xanthelasma seems hardly to be a true tumor but rather the result of local or widespread accumulation of lipoid substances in the cells of the reticulo-endothelial system. Aschoff says it is well known that the most varied lipoids can normally be stored

*From the Department of Surgery, the University of Rochester School of Medicine and Dentistry, and the Rochester General Hospital.

†This paper was to have been read by Dr. Phillips at the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minnesota, May 4, 5, 6, 1936, but his illness and death shortly before the meeting prevented the completion of the paper. It has been finished posthumously by Dr. John J. Morton.

in the spleen, especially in the reticulum cells, and writes of the accumulation of cholesterol esters in the various cells of the reticulo-endothelial system. Rowland[6] describes the xanthoma cell as a cell of the reticulo-endothelial system infiltrated with lipoids. King[7] believes that the foam cells, as well as the giant cells as they are found in "synovioma," are phagocytic cells. Ewing, in writing a description of a xanthoma of the orbit reported by Carey,[8] well expressed the confusion that exists when he admitted his indecision as to how to classify the tumor in question: "I have been seeing such orbital tumors as you send me for many years and have never known what to call them. I used the term endothelioma for many years and then discarded it. The origin from fat tissue has come next on my list. It should make the tumor a xanthoma or a liposarcoma. I do not think it is very malignant, but I have not followed the clinical history of my cases. The xanthomatous character of many cells is quite clear in your case. The yellow color favors an origin from fat tissue. Xanthosarcoma and liposarcoma mean practically the same thing." Garrett,[9] from Bloodgood's Laboratory, said they did not believe that there is anything specific about the greenish yellow color of the tumors of the xanthoma type, but that it is simply an indication of blood destruction, and quoted Mont Reid as believing that the foam cell is due to the absorption of cholesterin from the broken-down blood.

The foam cell occurs in inflammatory conditions and metabolic disturbances. The so-called xanthelasma of the eyelids and the subcutaneous nodules in diabetes belong in this classification. They are found in the walls of brain abscesses, the walls of a dermoid cyst, in chronic inflammations of the gallbladder, and in pyosalpinx. They are sometimes present with icterus and in the nephrosceleroses. They also occur in true neoplasms.

Newgrowths characterized by the presence of "foam" cells are rarely found, excepting those arising from the tendon sheaths, which have been variously called spindle-cell sarcoma, myeloid sarcoma, myeloid tumor, myeloma, myeloid endothelioma, myeloxanthoma, granuloma, giant cell tumor, giant cell sarcoma, and synovioma.

Wessén,[10] in reporting the pathologic description of an intrathoracic tumor removed in 1917 by Jacobaeus and Key, stated that he had not found in the literature accessible to him any case records of tumors of the size and location with a constant, not regressive layer of doubly refracting fatty substances in the tissue of the tumor. He referred to a case reported in 1913 by Dietrich[11] of a fibroxanthosarcoma of the retroperitoneal tissue, and a myxocholestolipoma, with the main localization in the retroperitoneal tissue, recorded by Proescher (quoted by Wessén). During June, 1923, before the American Surgical Association, Heuer[12] reported eight tumors of the thoracic wall, pleura, and

mediastinum. Included in this group was an intrathoracic tumor which he called a xanthoma or a xanthosarcoma, which he had operated upon during October, 1919. In a search of the literature he had not found the report of any similar tumors in that location. In 1928 Humiston and Piette[2] reported a xanthoma of the neck and said that in the recent literature, except for the tendon and joint xanthomata, they were able to find only three cases, reported by Smith[13] (tongue, labia minora, and parotid gland), one by Lutz (quoted by Smith) (mesentery), and one by Beitzke[14] (meninges). In 1924 Miller[15] reported the case of a woman with a breast tumor about the size of a grapefruit in which there was an area about the size of a hen's egg of brilliant color and containing typical foam cells of xanthoma tissue. Carey[8] in 1930 reported a xanthoma of the orbit.

Fig. 1.—Case 1. Roentgenogram showing a large intrathoracic tumor, occupying the upper two thirds of the left chest.

REPORT OF CASES

CASE 1.—Mrs. H. E. F., aged forty-four, a housewife, was first seen at the Rochester General Hospital on June 16, 1930. She was born in England and lived there for sixteen and a half years, and then moved to the United States. Her mother was living and well; her father died at twenty-eight years of age because of kidney trouble. She had four brothers living and well. One brother was killed during the war. Husband was living and well. There was no history of tuberculosis or other familial disease. During childhood she was never seriously ill, but had all of the usual childhood diseases. Since childhood she has had frequent attacks of bronchitis. When she was twenty-three years of age, she had a small egg-sized tumor removed from the right breast. This healed without complication, and there has been no recurrence. At the age of thirty-seven, she had a supravaginal hysterec-

tomy performed for fibroids of the uterus. Postoperative convalescence was uneventful. Menstrual periods began at eleven and a half years; they were never regular, varying from fourteen- to thirty-five-day interval, usually flowing five days. She has had no periods since her hysterectomy. She has never been pregnant.

Present illness—For five years previous to her admission she had a gradually increasing cough and during that time had occasional attacks of what her doctor thought were mild pneumonias. At these times the cough was very severe and productive, and the sputum was sometimes blood-streaked. In December, 1929, she had the last of these attacks, and since that time sputum has been less in amount. There was a dull pain in the left lower chest for about one year. In the four years prior to her admission she has had frequent attacks, which have been considered asthmatic attacks, during which respiration was of a wheezing character. Two years before entering the hospital she noticed a small soft mass in the right axilla. Because of this tumor, she consulted her physician, and interested by the history of pulmonary

Fig. 2.—Case 1. Lateral view of the intrathoracic tumor, showing the posterior position.

trouble, he made a roentgen ray of her chest and found that the upper left lung field was obliterated by a shadow of uniform density.

Examination revealed a well-developed and well-nourished woman of forty-four years of age. She was not acutely ill. There was no dyspnea or cyanosis. The pupils reacted to light and accommodation; they were regular and equal. There was no enophthalmos. The general physical examination was negative except for abnormalities found upon chest examination. In the right axilla, there was an egg-sized tumor immediately beneath the skin, soft and lobulated. This was undoubtedly a lipoma and was not productive of symptoms. The chest was symmetrical, no bulging or retraction of the intercostal spaces. The left chest showed a definite lagging during respiratory movements. The percussion note was slightly impaired over the left apex posteriorly, gradually increasing to flatness at the midscapular region. The breath and voice sounds were very distant over the area of impaired resonance. There were no râles. The right chest showed no abnormal signs. The heart was displaced toward the right. The right border by percussion was found

to be 3 cm. to the right of the sternum. The apex beat was best felt just to the left of the sternal edge. The heart sounds were regular and of good quality; there were no murmurs. The abdomen was negative except for a well-healed scar of a lower midline incision.

Roentgen ray examination of the chest—In the anteroposterior view, there was a large shadow in the left chest extending from the apex down to the level of the tenth rib near the spine and to the seventh rib at the lateral chest margin. The heart was somewhat displaced to the right. This mass was of uniform density. There was no evidence of erosion of vertebrae or ribs. The lateral view showed that the shadow originated in the posterior chest, and that there was a layer of normal lung between it and the anterior chest wall. Fluoroscopic examination corroborated the findings of the roentgen ray and demonstrated no pulsation in the shaded area (Figs. 1 and 2).

A study of the blood smear showed an eosinophilia of 1 per cent. There was no increase in the leucocytes. An intradermal skin test with echinococcus antigen

Fig. 3.—Case 1. Tumor after removal.

showed an immediate positive reaction which gradually disappeared at the end of twenty-four hours.

On June 17, 1930, under gas oxygen anesthesia (Dr. Sahler), supplemented with infiltration and nerve block with procaine hydrochloride, about five inches of the left sixth and seventh ribs were resected subperiosteally from the angles forward. The fourth rib was sectioned at the posterior end of the incision. The parietal pleura was incised for the full length of the incision. A large oval tumor was found occupying the upper portion of the thoracic cavity. It was firmly fixed to the posterior chest wall in the costovertebral gutter. The lung appeared normal and was loosely adherent to the tumor mass. These adhesions were readily separated by blunt dissection. The tumor did not extend into the mediastinum. It was separated from its posterior attachment by making an incision through the parietal pleura near the base of the tumor and with a sharp chisel cutting along the front of the ribs and corresponding transverse processes. During this procedure, there was brisk hemor-

rhage which could not be controlled until the tumor had been removed, when it was found that the bleeding was from two intercostal arteries that had been severed. It was impossible to repair the defect in the parietal pleura which remained after removal of the tumor. A closed drainage tube was inserted through the eighth intercostal space, and, with the lung fully expanded by positive pressure, the chest wall was closed in layers making an airtight closure.

The operation was well borne. Because of a moderate degree of shock associated with pallor, she was transfused with 650 c.c. of citrated blood a few hours after the operation.

The closed drainage tube which was inserted at the end of the operation failed to function properly, and on June 20 it was apparent from physical examination and roentgen ray study that there was present a large hydropneumothorax. This was associated with displacement of the heart to the right, considerable dyspnea, and slight cyanosis. The left chest was aspirated, and 500 c.c. of clear bloody serum and 530

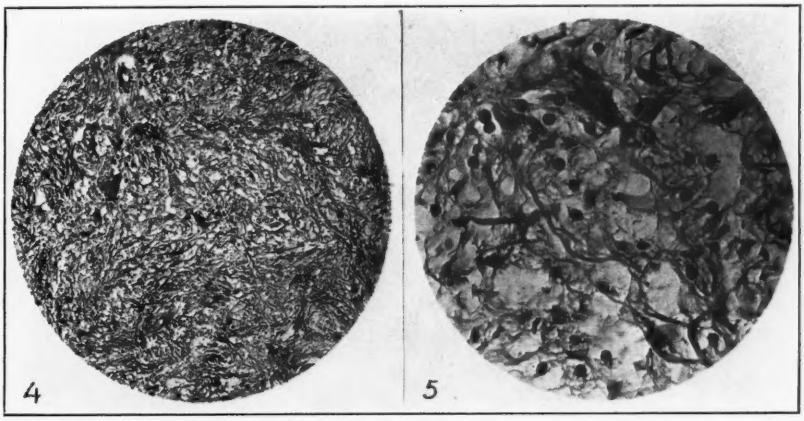


Fig. 4.—Case 1. Microphotograph of the tumor showing the characteristic xanthoma cells (foam cells). Low magnification.
Fig. 5.—Case 1. High power microphotograph.

c.c. of air were removed. Aspiration was again performed on June 26, and 400 c.c. of an amber-colored fluid and 400 c.c. of air were removed.

For the first few days postoperative, there was a moderate amount of subcutaneous emphysema of the chest wall in the neighborhood of the incision. About fourteen days after the operation, there was some purulent drainage from the posterior end of the incision. The incision up to this time had appeared clean. Two days later there was rather profuse drainage from the anterior end of the incision which lasted for two or three days. Considerable elevation of temperature persisted, going as high as 102° and not affected by the drainage of pus from the incision. On July 10 an aspiration of the left chest in the eighth interspace posteriorly recovered a small amount of thick pus of rather foul odor. Culture showed the pus to contain a nonhemolytic *Staphylococcus aureus*. On July 11 an empyema in the left chest was drained by subperiosteal resection of a portion of the eighth rib under local anesthesia. The lung was found to be loosely adherent to the chest wall. Everywhere on the pleural surfaces there was a shaggy, easily broken fibrinous layer, and scattered about were small puddles of pus of a disagreeable, nauseating odor.

The empyema was treated by dakinization, and the cavity slowly became smaller. Following the drainage of this infection, the patient's general condition improved rapidly. She was soon out of bed and walking about, and was discharged from the hospital on August 23, 1930.

Pathologic report from Dr. Gaspar: "An egg-shaped tumor received. It measures 17 × 11 × 8 cm. Surface is smooth. On one side there are numerous adhesions. Cut section shows an edematous tissue with numerous yellowish areas and some cystic cavities. The tumor is surrounded by a definite capsule. Histologically: The tumor is formed by connective tissue cells arranged in broad bundles. There are a few areas with peculiar giant cells, areas of edema, and large groups of xanthoma cells. These cells give the pale orange yellow color in the macroscopic specimens. Fat stain shows the abundant presence of fat. Mitotic figures are not found. With the Bielschowsky silver impregnation method no nerve fibers could be traced; however,

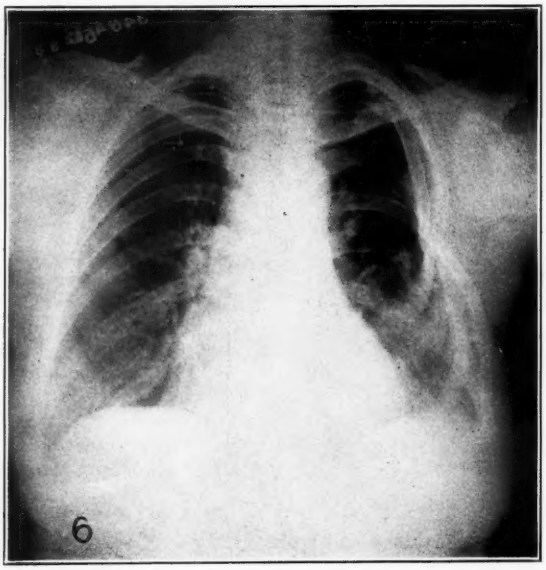


Fig. 6.—Case 1. Film taken March 30, 1936, nearly six years after removal of tumor. It shows operative deformity of ribs and no recurrence of tumor.

the location of the tumor suggests an origin from a peripheral nerve, possibly from the sympathetic plexus. The tumor is not malignant." (Figs. 3, 4, and 5.)

Dr. Ewing examined slides of this tumor and concluded that, although nerve elements could not be demonstrated, it originated from nerve tissue, either the sympathetic or the spinal nerve.

Diagnosis: Xanthofibroma.

Examination of this patient on March 30, 1936 (almost six years after removal of the intrathoracic tumor), did not reveal evidence of recurrence of the tumor. A chest roentgenogram revealed some deformity of the ribs on the operated side, but there was no evidence of recurrent newgrowth (Fig. 6).

CASE 2.—Mrs. C. J., a housewife, aged forty-seven, was first admitted to the Strong Memorial Hospital on October 31, 1933, complaining of pain in the right

axillary region. An "itching" pain in the right axilla was first noticed twenty-eight years previous; it came on during menses. It has recurred at menses, radiating to the right nipple and rendering the entire right breast tender. Pain was not influenced by respiration or motion of right shoulder. There was no cough, expectoration, or hemoptysis, and no recent weight loss. Several weeks before admission, her husband was found to have tuberculosis. Patient was examined at Monroe County Tuberculosis Sanatorium as a contact. No tuberculosis was found, but x-ray showed an area of increased density 4.5 × 10 cm. between the third and seventh ribs, right. The chest was aspirated; no fluid, but definite resistance was felt. On admission temperature was 37°, pulse 80, respiration 22, and blood pressure 142/80.

The chest was symmetrical, expanded equally. In the lungs the percussion note was resonant throughout except for some impairment along lateral aspect of right

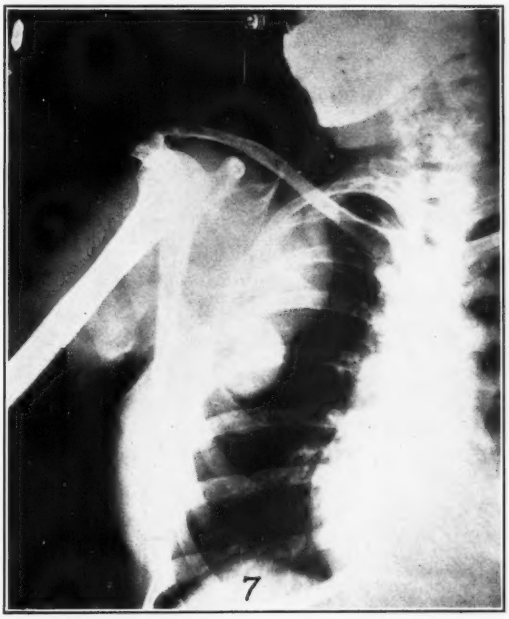


Fig. 7.—Case 2. Roentgenogram showing tumor of uniform density in the anterolateral right chest.

anterior chest, with partial suppression of breath sounds in the same area. Between the third and fourth right anterior ribs in the region of the anterior axillary line there was a palpable smooth oblong mass about 3 × 4 cm., fixed; overlying skin not attached. Heart was not enlarged; soft systolic blow localized at apex; rhythm regular.

Urinalysis—negative. Wassermann—negative.

Roentgen ray examination of chest on September 13, 1933, disclosed a dense tumor mass in the anterior chest wall measuring 11 × 4 cm., sharply circumscribed and composed of two definite lobes. "It appears to lie in the soft tissues of the anterior chest wall without involving the ribs." (Fig. 7.)

W.B.C.—13,333. Hemoglobin—102 per cent.

November 7, 1933—Fluoroscopic examination was made. The tumor did not move with the scapula. Chief motion associated with respiration: moved one interspace

during deep inspiration. It was difficult to determine whether the tumor was inside or outside of the chest wall; it was definitely not attached to the ribs.

November 8, 1933—Biopsy of extrathoracic portion of tumor. Firm, nodular, yellowish white in color, about 3 × 5 cm., between second and third ribs, not adherent to ribs. A large portion of tumor could be seen through the pleura extending up toward the apex. Projecting portion removed. Microscopic—Benign; chiefly fibrous tissue. "Moderately dense, fibrous tissue arranged in interlacing bundles and is quite cellular. There are more vesicular areas between bundles where what ap-

Fig. 8.—Case 2. Tumor after removal.

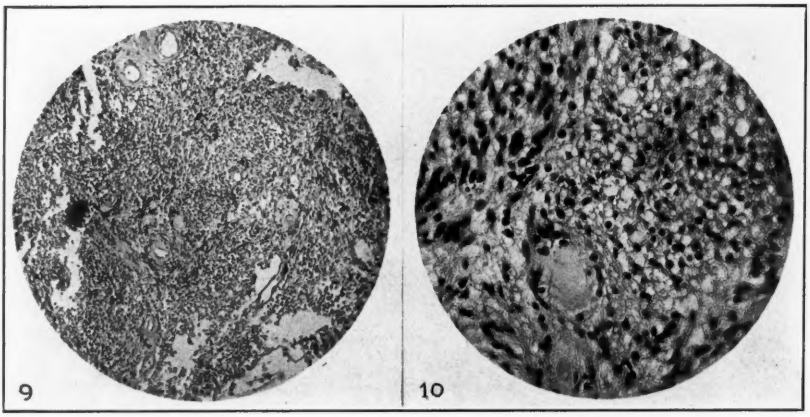


Fig. 9.—Case 2. Low power microphotograph showing the xanthoma cells (foam cells).

Fig. 10.—Case 2. Higher magnification of the same section.

pears to be cross-section of nerve fibers may be seen; tangential sections of these fibers are more rarely encountered. No ganglion cells are made out. Tumor is moderately vascular. A few islands of lymphoid tissue are seen. A loose fibrous capsule may be seen on uncut surface."

Readmitted January 24, 1934, for removal of intrathoracic tumor.

Operation January 25, 1934. Gas oxygen anesthesia plus local. Incision encircling outer and inferior margins of the breast. Breast reflected up and inward. Pectoralis major displaced upward; some fibers cut. Pectoralis minor fibers divided.

Third rib resected; in anterior portion it was normal; but over tumor it was thinned out and ridged. Parietal pleura at tumor so blended as to be unrecognizable; a portion of it was resected with tumor. Anterior to the tumor it was thin and transparent. Tumor freed with finger dissection; lower end turned into incision and slowly delivered and removed (Fig. 8).

Pathologic study—Diagnosis: Xanthofibroma of thorax. Gross: A pale yellow sausage-shaped tumor 15 cm. long and 4 cm. in diameter. The surface was slightly nodular and irregular. On cut section the tissue was pale and cellular with many deep orange-colored masses imbedded in it. Several small cystic areas were encountered. Two small sections of ribs showed definite pressure erosion. Microscopic:

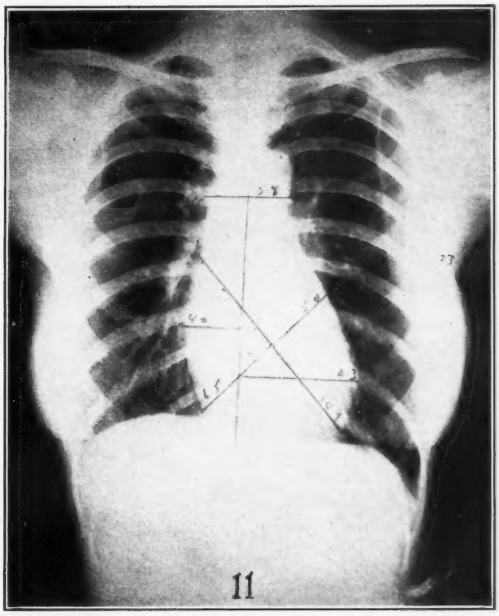


Fig. 11.—Case 2. Follow up roentgenograph two years and three months after removal of the intrathoracic tumor.

The tumor showed dense hyalinized fibrous tissue at its periphery. Toward the center the stroma was loose, with areas of colloid alternating with foamy areas of small vacuoled cells with tiny nuclei. A few broad bundles of fibrous tissue (and some large giant cells) were present. No ganglion cells were encountered. The tumor was quite cellular in areas, with fibroblasts and fibrillary processes very distinct (Figs. 9 and 10).

Postoperative convalescence was uneventful.

Follow-up examination on March 31, 1936: The patient has had no symptoms referable to the chest. There were no abnormal findings upon examination, and the x-ray film made on March 30, 1936, did not show recurrence of the tumor (Fig. 11). Although the Pathological Department thought this tumor showed early malignant degenerative changes, it is probable that it was nonmalignant.

CASES REPORTED IN THE LITERATURE

JACOBAEUS AND KEY'S CASE[16] (cellular fibroxantholeiomyoma) (1921).—Woman, aged twenty-eight. Previously healthy, she began in April, 1916, to suffer from

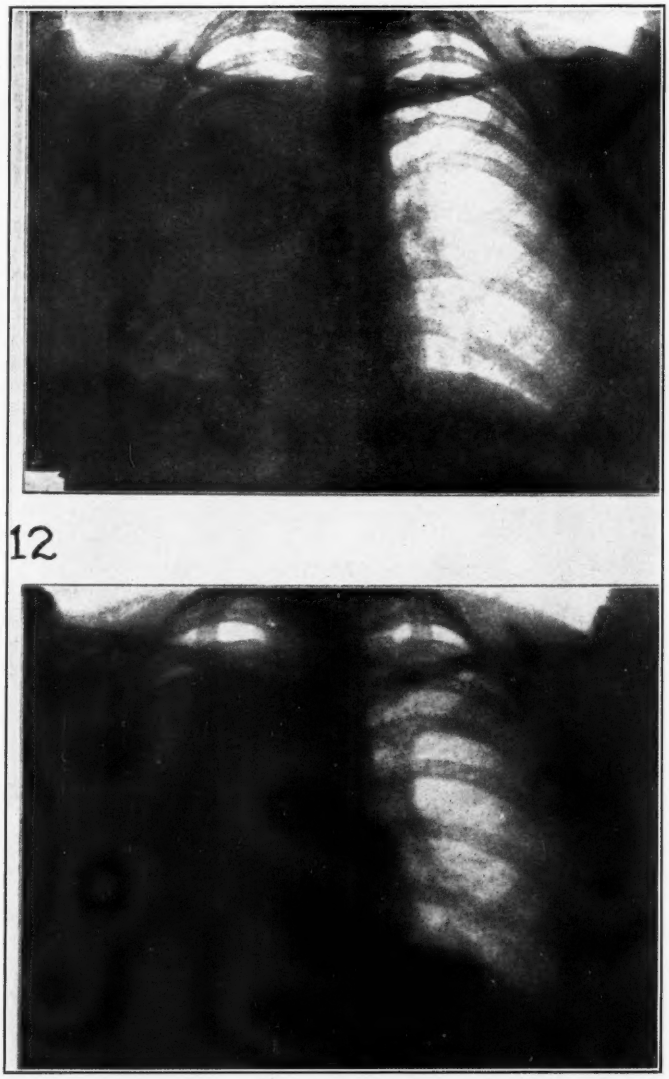


Fig. 12.—Jacobaeus and Key's Case. Roentgenogram showing large intrathoracic tumor occupying the greater part of the left chest.

paroxysmal cough. At this time her children were ill with pertussis, and at first her cough was attributed to the same cause. The cough was accompanied by a diffuse

bronchitis with râles, more marked on the left and associated with diminished respiratory sounds on the left side. Signs of an exudate in the left pleura appeared. During the summer the exudate disappeared, but weakened respiratory sounds persisted. During the autumn her troubles increased. The patient suffered from shortness of breath, especially in mounting the stairs, and she had a hacking cough. On December 22, 1916, she developed pain in the cardiac region and in the left side which lasted about an hour. On the following day examination revealed an exudate in the left pleural cavity. There was no fever. The exudate increased and reached the level of the spine of the scapula. It gradually sank again, but dullness remained with great weakening of the respiratory signs, and some shifting of the heart to the right. An intrathoracic tumor was suspected, and she was sent to Professor Forssell for roentgen ray examination. Corresponding to the lower part of the upper lobe of the left lung, there was a rounded compact tumor with sharp linear outline, reaching from the first rib in the front down to the sixth rib and outward nearly up to the inner surface of the thoracic wall. The tumor lay nearer the back (Fig. 12). No positive changes in the right lung. The left diaphragm had somewhat less motion than the right, and in the middle the diaphragm outline seemed to be drawn up in a small point (left from a former attack of pleurisy).

The patient was admitted to the Maria Hospital on January 19, 1917. At this time she complained that she easily became short of breath. She was moderately obese. There was no cyanosis. Examination of the thorax revealed no abnormal vein design. Both sides of the thorax appeared alike. There was marked dullness in the left back from the spine of the scapula to the base. Breath sounds were markedly weakened on the left side. Auscultation of the heart was negative. The right border was at the right sternal edge or possibly one centimeter to the right of it. To the left heart limit was not definable. Aspiration of the left pleural cavity recovered 300 c.c. of sanguineous fluid. Examination of the fluid by Professor Quensel showed red blood cells in ample quantity, polynuclear leucocytes and mainly mononuclear cells varying in size from nine to twelve microns up to thirty to thirty-three microns. The larger cells were often in groups together, and often vacuolized and contained fat in more or less ample quantity. "The cytological appearance corresponds with that one usually sees in pleural fluid when malignant tumor is met with." Thoracoscopic examination was performed on January 23, and the left pleural cavity was seen to be filled in the larger part by an oval, grayish tumor, the surface of which seemed on the whole to be smooth, with here and there raised lines. At one place, there was a cyst about the size of a bean. The lung appeared normal. The surface of the diaphragm and the parietal pleura showed a group of pea-sized, dark red efflorescences without any visible vessel design.

Operation was performed on January 25 under general anesthesia, with the Elsberg's insufflation apparatus. Sixteen centimeters of the sixth rib were resected from the angle forward to the midaxillary line. The pleura was opened along the whole length of the incision. A fairly plentiful amount of hemorrhagic fluid was evacuated from the pleural cavity. In the posterior part of the pleural cavity, there was a tumor scarcely the size of a child's head, with smooth white surface; it was of elastic consistence. The tumor was fixed to the chest wall over an area as large as the hand. The area of fixation began on the lateral side of the vertebrae and ended some centimeters medial to the angles of the ribs. The descending aorta was felt just medial to the tumor, but apparently was not adherent to it. There were scattered small deposits of fibrin upon the pleura. Exposure was insufficient and sections were removed from the fifth, seventh, and eighth ribs, which gave sufficient space. The tumor was removed, leaving a defect in the parietal pleura about the size of a child's hand, which could not be covered. The incision was closed completely.

Following the operation, the patient was in considerable shock. During the afternoon of the day of the operation, the right radial pulse was not perceptible and the left slightly perceptible. By evening the pulse had improved so that the right was also perceptible. Convalescence was complicated by a pleural effusion causing cyanosis. On January 29, 1,400 c.c. of slightly bloody fluid were removed, and

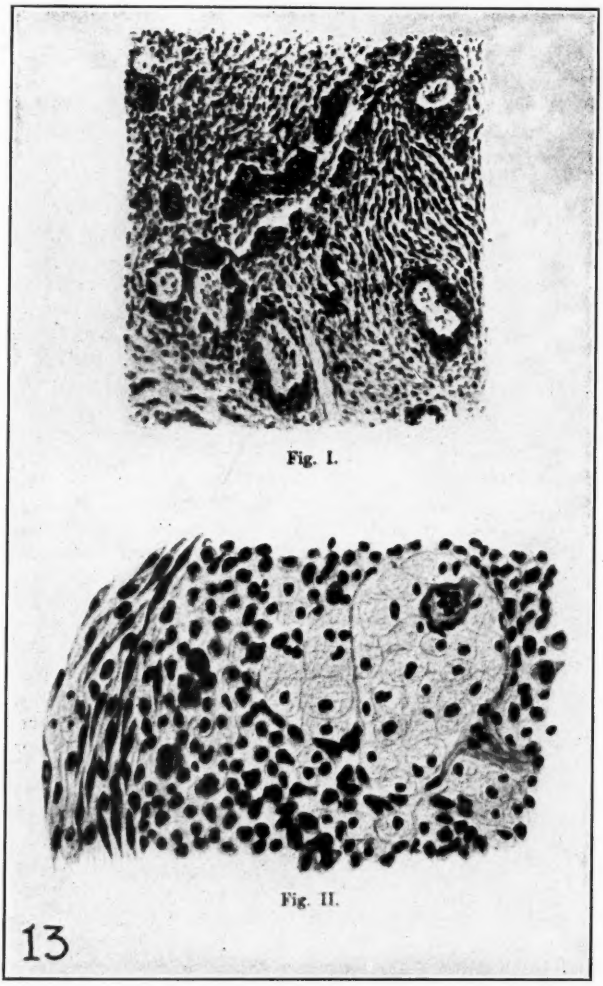


Fig. 13.—Jacobaeus and Key's Case. Low and high power magnification microphotograph.

again on January 30 another 300 c.c. were aspirated. On February 2 only a few cubic centimeters of clear fluid were recovered. Again on February 10, because of dyspnea and the physical signs of fluid, the chest was aspirated and 300 c.c. of somewhat bloody fluid were recovered. The patient gradually and completely recovered. On February 1, 1921, she was still reported as being in sound health.

Pathologic report (Wessén): The tumor, which weighed 515 gr., was fixed in formalin solution; it had a rounded, oblong form and measured 13 × 9.5 × 6 cm. The fresh tumor was of a somewhat elastic consistency and had a loose pleural covering, except at one side, corresponding to the attachment to the thorax, about 12 cm. long and 6 cm. broad. The pleura-covered surface was pale and generally smooth, although in several places it showed small fibrous thickenings. The freshly cut one showed a surface of a peculiar yellowish color with scattered small grayish red strata and spots. The tumor contained two cysts, nearly as big as walnuts, and two smaller ones, all sharply defined, with a smooth, partly injected wall. On section the parenchyma was felt to be of a generally rather firm, elastic consistency, with still firmer parts at isolated places on its periphery.

Microscopic examination: Frozen sections of the tumor, which had been exposed to a formol hardening and stained with scharlach red hematoxylin, showed a generally uniform structure: a fibromatous ground substance, rich in cells, with fibrillae, spreading smoothly and loosely with an abundant stratification of yellowish red-stained, fatty substance in the form of crystals and drops. The localization of big drops and crystalline hard spots around the smaller vessels, as well as inside the numerous round or oval cell groups, is especially remarkable, above all in the central parts of the tumor. The crystals are generally formed like needles with broken ends; sometimes they swell, forming rhombic disks. They are of different sizes and lie isolated or concentrated in bundles. The crystals, on staining with scharlach red or sudan III, appear light yellowish red; the drops showed a more distinctly red color. Between the single threads and in the cells of connective tissue, the fat appears partly as small laminae and partly, as a rule, as drops. On examining with a polarization microscope a nonstained section of the tumor, preserved in formalin, one finds that nearly all these drops show a double refraction, but single ones are isotropical. The crystalline masses all show double refraction; this double refraction varies in the dark optic field in such a way that the luminous drops are partly divided into four fields by the black transverse axes and partly diminish into mere lustrous traces in the outlines of the drops. When the preparation is heated, the crystals melt and change into drops; anisotropine, which only then disappears, reappears rather prettily on freezing. Osmium stains the drops as well as the crystals black; nile blue sulfate stains the drops of fat in the connective tissue rosy; the larger drops, especially those in the thick concentrations, violet blue, while the crystalline masses assume a pale light red stain. Sections, exposed to chrome for twenty-four hours, stained according to Lorrain-Smith-Dietrich's method, show isolated black-colored drops (isotrop); the double refracting drops remain colorless or show a slightly stained outline; the crystalline masses assume only a pale grayish shade.

Hematoxylin—von Gieson's stain—characterizes the tissue of the tumor as possessing a celliferous, generally loose, fibromatous structure which, owing to the abundance of cells and their polymorphism, in many places has a certain sarcomatous character. Numerous strata of smooth muscular tissue run through the tissue of the tumor; this seems especially to be the case in the part nearest to the attachment of the tumor, and, as a rule, in its outer layer. The connective tissue shows in many parts a very marked hyalinization, above all in those vessels which are surrounded by masses of fat. In places there also appear spots of rich cell abundance generally composed of large cells of the fibroblast type without any definite depositing, gradually passing over into isolated, big, pale, rounded cells with a granular, finely vacuolized protoplasma and a comparatively big nucleus, the xanthom cells (Schaumzellen), or into small groups of such cell layers. These cells change in their turn into large complexes with a definite structure of xanthom cells, where the cells seem to appear without contour in many places. These xanthom cell complexes correspond in the fat-stained preparations to the sections where the big, palely

stained crystalline masses are situated. Isolated xanthom cells are also found here and there between the threads of the connective tissue and those of the muscular tissue. In the inner parts of the tumor the xanthom cells are as a rule con-

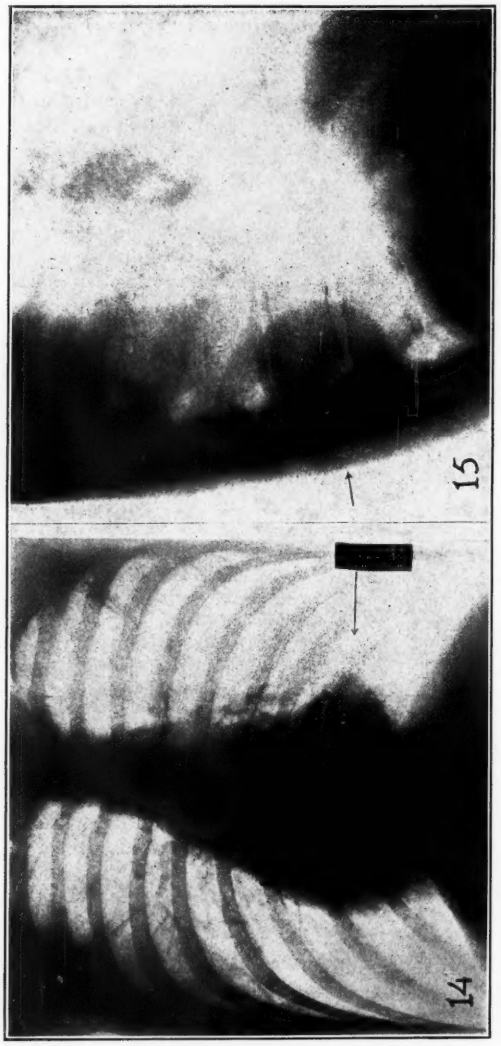


Fig. 14.—Heuer's Case. Roentgenograph showing small rounded shadow in right lower chest.
Fig. 15.—Heuer's Case. Lateral view showing posterior location.

centrated around the vessels, corresponding to the layers of fatty substances in the scharlach-red-stained preparations (Fig. 13).

HEUER'S CASE[16] (xanthoma or xanthosarcoma) (1923).—The patient, a physician, aged twenty-nine years, had for fourteen years pain of a cramplike nature in the right lower quadrant. The pain was not associated with fever or nausea; nevertheless, in 1911, on the advice of his physician, he had his appendix removed. The ap-

pendix was normal and its removal failed to relieve his pain. During his service with the army in France, the pain became more severe and he was repeatedly examined. A relaxed inguinal ring with an impulse was noted, and a hernia operation was suggested but refused. Cystoscopic examination, catheterization of the ureters,

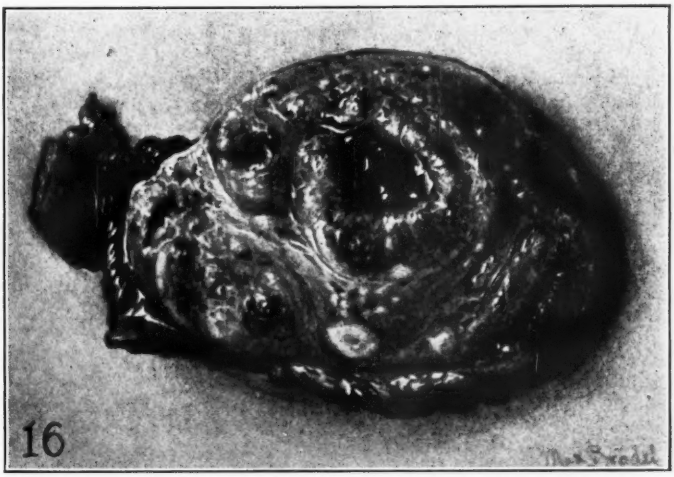


Fig. 16.—Heuer's Case. Tumor after removal.

Fig. 17.—Heuer's Case. Microphotograph showing xanthoma cells.

and thorium injections of the renal pelvis with roentgen ray examinations failed to discover any abnormality. Roentgen rays of the gastrointestinal tract were negative. He continued to have pain in his lower right quadrant, but in addition began to have severe pain in his back, which was called lumbago. In November, 1918, he had influenza, and a roentgen ray of his chest showed a mass behind and to the right

of his heart shadow. Subsequent roentgenograms confirmed this finding. He was explored with a needle, but the exploration was negative. On his return from France he was given radium treatment in Baltimore, but without benefit. His physical examination was negative except for the tumor shown in the x-rays (Figs. 14 and 15).

The operation was performed October 15, 1919. A long incision was made over and parallel with the ninth rib. The proximal six inches of this rib were resected, and the parietal pleura stripped from the thoracic wall over a wide area. Without opening the pleural cavity the tumor was exposed. It was larger than a hen's egg, lay against the bodies of the vertebrae, and seemed attached to the tenth rib (Figs. 16 and 17). The pleura was readily separated from it. Having freed the tumor down to its apparent attachment to the ribs, portions of the tenth and eleventh ribs were resected and removed with the attached tumor. The operation was completed without at any time opening the pleura. The wound was closed without drainage.

Postoperative convalescence was uneventful with the exception that the extrapleural space filled with fluid, which, however, subsequently disappeared. The wound healed *per primam*. The patient is entirely well and free from pain four years after operation.

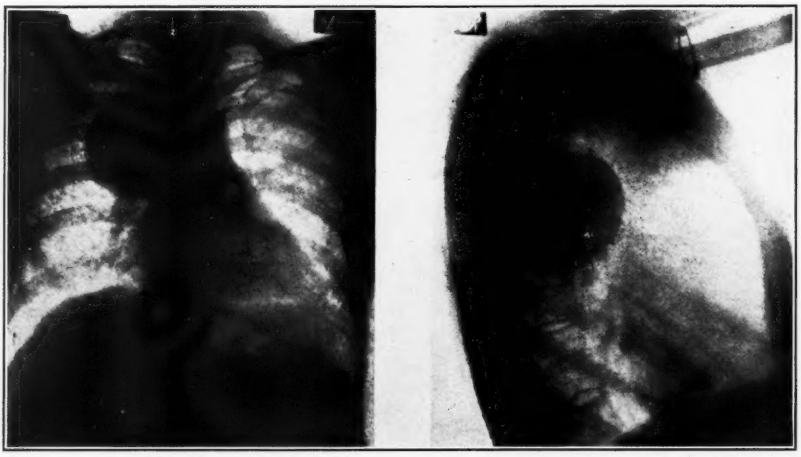


Fig. 18.—Edwards' Case. Anteroposterior and lateral roentgenograms showing small, round tumor in the right posterior chest.

EDWARDS' CASE[17] (lipofibrosarcoma) (1927).—Mrs. L. D., aged forty-seven, was admitted to the Westminster Hospital on April 16, 1926. There were no points of any significance in her past history except that she had had a cough following dry pleurisy four years previously. The present trouble commenced with pain in the right chest ten months before. It was becoming gradually worse and more continuous, though it was not enough to interfere with sleep. Examination gave evidence of a patch of dullness and diminished breath sounds over the right chest posteriorly in the region of the spine of the scapula and below. Roentgen ray examination showed an oval shadow in the upper part of the right chest just external to the vertebral column and overlying the angles of the fifth, sixth, and seventh ribs at their posterior ends. A lateral view showed the shadow to be within the chest cavity (Fig. 18). Artificial pneumothorax (April 22) was performed and the lung partially collapsed. A skiagram showed the edge of the tumor beyond the lung edge above, thus proving it was extrapulmonary. Thoracoscopy (April 26) was performed and the instrument introduced below. The tumor could not be directly seen

Fig. 19.—Edwards' Case. Tumor after removal.

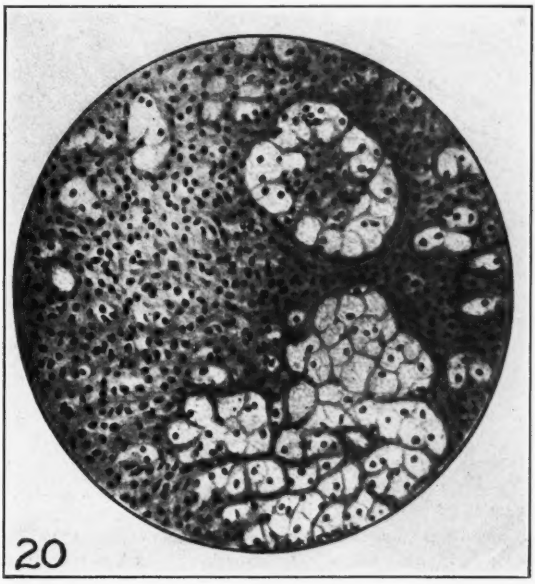


Fig. 20.—Edwards' Case. Microphotograph showing groups of tumor cells.

from this position, but a definite localized bulging of the lung was visible. The reason for this was disclosed at operation, where the lower part of the tumor was found covered by adherent lung.

Operation (April 29).—Intratracheal anesthesia (Dr. Magill). The incision was made in the seventh intercostal space from the angles of the ribs forward to the

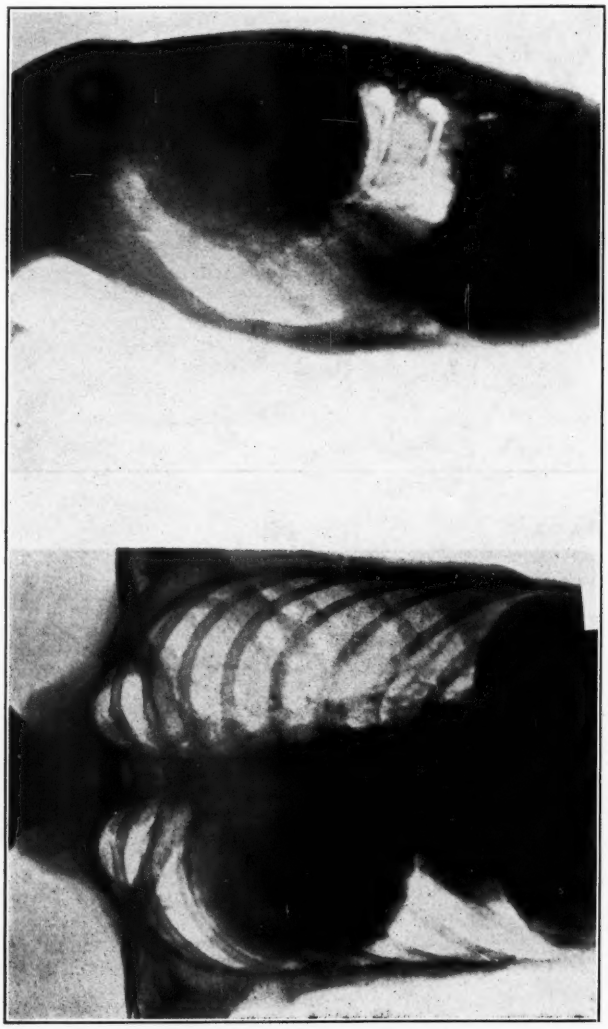


Fig. 22.

Fig. 21.

Figs. 21 and 22.—Hess and Morris' Case. Anteroposterior and lateral roentgenograms showing large intrathoracic tumor in posterior left chest.

axillary line. The pleura was opened, and the tumor seen lying in the costovertebral groove as shown in the skiagrams. The seventh and sixth ribs were subperiosteally divided, but no portion of bone was removed. This gave considerable increase of exposure without any division of the soft tissues. The adherent portion of the lung was separated from the remainder of the lobe, and the raw area in the latter was

sutured. The pleura on the outer side of the tumor was incised, and the tumor removed from its attachment to the fifth rib together with the adjacent periosteum. The pleural cavity was mopped dry and the chest wall sutured in layers, the lung being inflated by positive endotracheal pressure before tying the last suture. No peri- or percostal suture was inserted, but the chest wall was firmly strapped over the dressings by overlapping strips in the manner adopted for fractured ribs. Convalescence was rapid and uninterrupted except for the withdrawal of 5 oz. of blood-stained effusion on the fifth day. The patient was sitting up out of bed on the thirteenth day.

Pathologic report (taken from the specimen in the R.C.S. Museum): The tumor is nearly circular in outline, 2½ inches in diameter, smooth on the surface, except for a portion of adherent lung, and surrounded by a delicate fibrous capsule. In the section it is seen to consist of two types of tissue: a central dense mass having the appearance of hyaline fibrous tissue, and irregular, opaque, buff-colored areas of softer consistency, which are in places divided up by narrow hyaline tracts (Fig. 19).

Microscopic examination: This showed the growth to be a lipofibrosarcoma. It consisted partly of areas of closely packed spindle cells, partly of areas of small polyhedral cells, and extensive tracts of fat cells of an embryonic type, the fat being in the form of minute granules within the protoplasm. In the central parts of the tumor there was a large admixture of fibrous tissue (Fig. 20).

HESS AND MORRIS' CASE[18] (subpleural fibrolipoma) (1926).—This case is included as probably belonging to this group of tumors, although the pathologic diagnosis was fibrolipoma and not fibroxanthoma. According to Dr. Ewing, however, the two terms mean practically the same thing. The roentgen ray appearance of the tumor and the general description make it seem identical with the other cases that have been described. Unfortunately, microscopic slides or microphotographs are not available.[19]

Miss G., forty-nine years old, white, weight about 90 pounds, matron in City Comfort Station. Shortness of breath upon slight exertion or when in a crowd came on after influenza in January, 1921. Pleurisy noted in left side in March, 1924; sick about ten days. Again in June, 1924, she was sick in bed one week with left pleurisy. No weight loss; appetite poor; tires easily. No tuberculosis or cancer in family. Wassermann negative. She was poorly nourished and had a sallow, muddy complexion. She was slow in speech and movement.

Roentgen ray, July 3, 1924, disclosed a dense, sharply outlined spherical mass, about 12.5 cm. in diameter, located at the base of the left posterior chest, between the fifth rib and eleventh interspace. It showed no evidence of invading the remaining parts of the chest, and appeared to be arising from the left side of the vertebral border. Fluoroscopically it was fixed. It did not move with respiration, did not pulsate, and did not displace the heart (Figs. 21 and 22).

Conclusion—A cyst of the left chest attached to the left side of the spinal column. Reexamination July 14, 1924, and January 17, 1925, showed no change.

Operation (Dr. F. S. Morris[19]).—A U-shaped flap of skin, underlying tissue, and four ribs were reflected backward, breaking the ribs posteriorly. This gave an excellent exposure of the whole tumor, and there was very little embarrassment of respiration. The tumor was behind the pleura and close to the aorta. An incision was made near its base through the pleural membrane on the aortic side. The tumor rolled freely laterally and was attached only by soft areolar tissue and two blood vessels of moderate size. A second incision was made on the outer aspect, the vessels clamped and the tumor removed. The raw surface posteriorly was then covered by a running suture approximating the cut edges of the pleura. A small drainage tube was placed beneath the pleura at the lower angle, and a second one

in the pleural cavity in the lower anterior margin of the flap. The soft tissue and ribs were then sutured into place with chromic catgut and silkworm gut.

Pathologic diagnosis made by Dr. J. G. Wurtz, pathologist, was subpleural fibrolipoma. This diagnosis was confirmed by members of the Chemical Pathological Society of Pittsburgh.

Six months after operation, a roentgen ray showed resection of the sixth, seventh, eighth, and ninth ribs at posterior angle on the left side, with no evidence of the previously existing tumor. Lungs and mediastinum were normal. No gain in weight and general appearance unchanged.

SUMMARY

This group of intrathoracic tumors, though variously named, have many characteristics in common. Differing from the common xanthomas which are not firmly encapsulated or separated from the surrounding structures, the intrathoracic tumors here described are surrounded by a firm capsule and separated from the surrounding tissues. They are solid, firm oval tumors varying in size from "larger than a hen's egg" (Heuer) to the size of a child's head (Jacobeus and Key, Phillips). The origin is from the costovertebral groove. The surface is smooth except where attached to the thoracic wall. The freshly cut surfaces of the tumors show a peculiar yellowish color from which the name xanthoma is derived. Although the descriptive names of these tumors include the word sarcoma, they are, apparently, nonmalignant tumors. It would seem advisable to discontinue referring to them as sarcomata. King in a paper concerning the pathology of tumors of tendon sheaths suggests tenosynovioma as a properly descriptive name and says "The sarcomata have in recent years gradually been reduced from a numerically impregnable position, by segregation of the sarcomata of the testis, retroperitoneal tissues, mediastinum, etc., until now, with the exception of the osteogenic sarcomata, they comprise a decimated band which is viewed with considerable suspicion."

The structure of xanthoma is characterized by the great abundance of lipoid globules in large swollen polyhedral cells and smaller spindle-shaped cells. This material is an orange yellow lipochrome, more of it stained by sudan III than by osmic acid. The globules are doubly refractive and of myelin nature or probably a cholesterin fatty acid ester. The central cells may become greatly distended with this material while the peripheral cells may show the spindle shape of growing fibroblasts.

REFERENCES

1. Ewing, James: Neoplastic Diseases, Philadelphia, W. B. Saunders Co.
   Virchow
   Hallopean and Torok } Quoted by Ewing.
2. Humiston, C. E., and Piette, E. C.: Ann. Surg. **88**: 124, 1928.
   Wile, U. J., Eckstein, H. C., and Curtis, A. C.: Arch. Dermat. & Syph. **19**: 35, 1929.

3. Mason, Michael L., and Woolston, W. H.: Arch. Surg. **15**: 499, 1927.
4. Buxton, St. J. D.: Brit. J. Surg. **10**: 469, 1923.
5. MacCallum: A Textbook of Pathology, ed. 4, Philadelphia, 1928, W. B. Saunders Co.
6. Rowland, R. S.: Arch. Int. Med. **42**: 611, 1928.
7. King, E. S. J.: Brit. J. Surg. **18**: 594, 1931.
8. Cary, E. H.: J. A. M. A. **95**: 1806, 1930.
9. Garrett, Charles A.: Arch. Surg. **8**: 890, 1924.
10. Wessén, N.: Acta. chir. Scandinav. **53**: 621, 1921.
Dietrich, Proescher, Lubarsch } Quoted by Wessén.
11. Dietrich, A.: Virchows Arch. **212**: 119, 1913.
12. Heuer, George J.: Ann. Surg. **79**: 670, 1924.
13. Smith, Carroll: Surg., Gynec. & Obst. **14**: 551, 1912.
Lutz—Quoted by Smith.
14. Beitzke, H.: Zieglers Beitraege **69**: 400, 1921.
15. Miller, Edwin M.: Ann. Surg. **80**: 256, 1924.
16. Jacobaeus, H. C., and Key, E.: Acta. chir. Scandinav. **53**: 573, 1921.
17. Edwards, A. Tudor: Brit. J. Surg. **14**: 607, 1927.
18. Hess, George H.: Radiology **6**: 525, 1926.
19. Personal communication from Dr. F. S. Morris.

AN ADJUSTABLE CONTINUOUS WATER SUCTION APPARATUS FOR ASPIRATING CAVITIES

WALTER S. GLAZER, A.B., M.S., M.D.
DETROIT, MICH.

IN A RECENT issue of this publication[1] I described an apparatus for the continuous aspiration of a cavity at a fixed negative pressure. While this apparatus functioned very well, and could readily be made at a small cost by almost anyone, it soon became apparent that it would

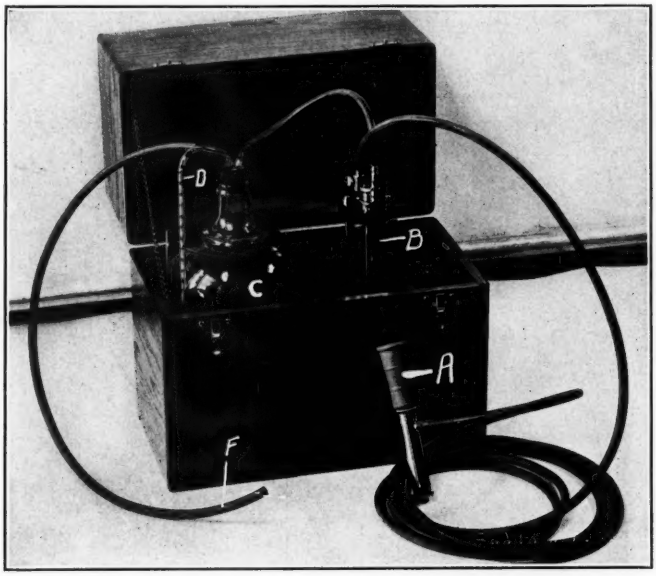


Fig. 1.

be much more desirable to have a complete unit which could be kept assembled and ready for instantaneous use. In the apparatus described there was also occasional trouble from leaking corks. To meet these desires and correct these faults, the apparatus here described was developed.

This apparatus works on exactly the same principle as the one previously described, but in operation it has been found to be much more practical. It is contained in a carrying case of about 15 x 15 x 7 inches, which makes it very practical from the standpoint of storage, as well

as not being in the way, or alarming the patient when in operation. Fig. 1 shows the complete outfit in its case, ready for use. Fig. 2 demonstrates the details of the regulator.

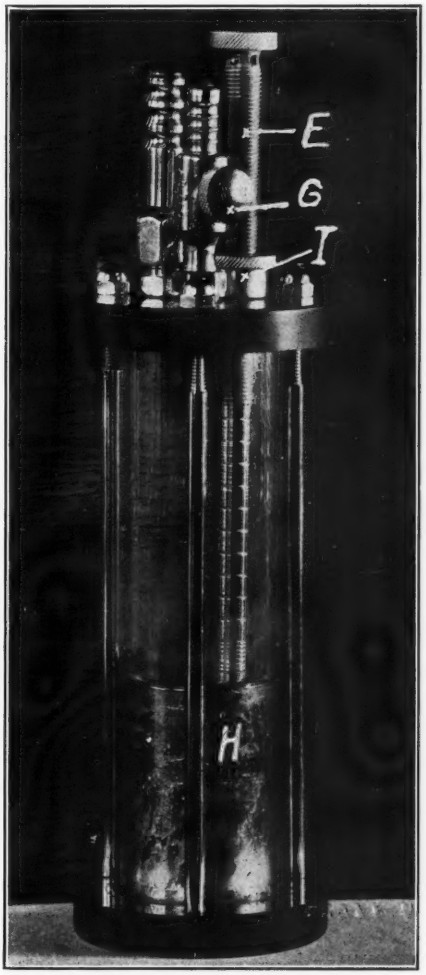


Fig. 2.

The source of the suction is the water filter pump, *A*, which can be attached to any nearby water faucet. This is then attached to the regulator (*B* and Fig. 2), which can be set at any degree of negative pressure from –5 to –65 mm. Hg. Through this the pressure in the collecting jug, *C*, is adjusted and kept constant, thus giving a uniform pull upon the cavity to be aspirated. In the apparatus as pictured, the col-

lecting jug is fitted with a manometer, *D*, which gives visual evidence of the degree and uniformity of the suction. This may or may not be used, depending upon the individual desire, as the adjusting tube, *E*, of the regulator is calibrated, and once set cannot change. Tube *F* leads from the collecting jug to the cavity to be aspirated.

Once set in operation, the outfit will continue to function without attention or adjustment for weeks at a time, excepting to empty the collecting jug as it becomes filled with aspirated fluid. As the jug has a capacity of one gallon, it is very rare that it must be emptied more than once a day.

The regulator is fitted with a safety valve, *G*, which prevents the flow of water from the pump to the regulator or collecting jug and also prevents a sudden loss of vacuum in the collecting jug when the water is turned off. It also is fitted with an adjusting tube, *E*, the lower end of which is calibrated from 5 to 65 mm. Hg. The calibrations up to 15 are 1 mm. apart and from there to 65 are 5 mm. apart. This tube is screwed up or down till the mark indicating the desired pressure is at the surface of the mercury, *H*, in the regulator and it is then secured in position by tightening the lock nut, *I*. This is the only movable part of the apparatus and consequently the only adjustment to be made. It removes all uncertainty as to the pressure, and once set cannot move unless intentionally done.

The apparatus as here pictured and described has been evolved over a period of many months, any defects corrected as they became apparent and refinements made as they suggested themselves. It is felt that in the present form it is entirely satisfactory and functions as perfectly as any machine can.

A more detailed description of the mechanics of its operation will be found in the publication previously referred to.

While the apparatus has been used locally with the greatest satisfaction in thoracic surgery, it has been found to be equally efficient in any other field where a constant suction is desired. In the field of thoracic surgery the pressure range most used is from –10 to –30 mm. Hg; in urological work –10 to –20 mm. Hg, and for use with a Levine tube –40 to –65 mm. Hg are used.

REFERENCE

1. Glazer, Walter S.: A Continuous Automatic Water Suction Apparatus for Aspirating Cavities, J. THORACIC SURG. 5: 557, 1936.

AIRTIGHT CLOSURE OF THE CHEST FOLLOWING PNEUMOLYSIS*

The Use of a Muscle Plug

Richard H. Overholt, M.D.
Boston, Mass.

THE prevention of the loss of air from the pleural cavity following internal pneumolysis may be of vital importance in the determination of a satisfactory result. The reexpansion of the lung, partially

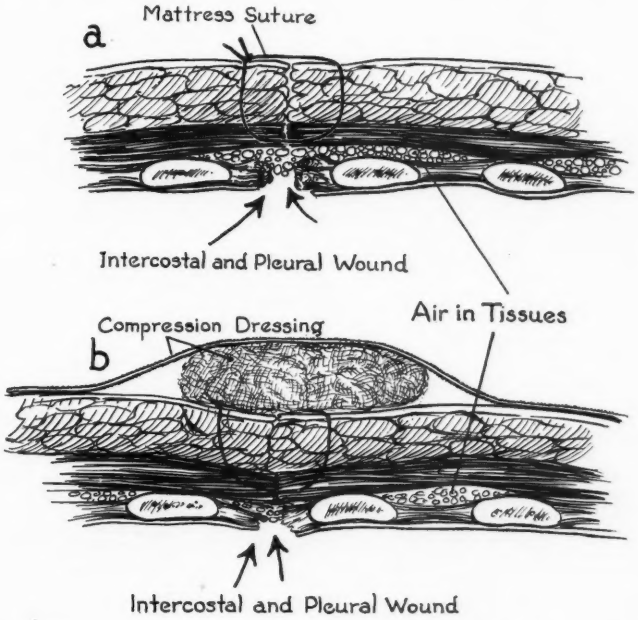


Fig. 1.—Illustration showing inadequacy of deep suture in the soft tissues of the chest wall in trocar wounds. The intercostal and pleural portions of the wound are protected by the ribs. Note emphysema of tissues. Note effect of compression dressing. The pleural opening is not closed and air may escape when coughing raises the intrapleural pressure.

or completely, as a consequence of an escape of air, may permit the reattachment of stumps of adhesions, the formation of new adhesions, or what is more unfortunate, the loss of the pneumothorax space to such an extent that its abandonment is forced. In addition, emphysema is undesirable from the standpoint of the comfort of the patient. These complications are more likely to occur, first, in patients who have violent

*From the Department of Thoracic Surgery, the Lahey Clinic, Boston, Mass.

coughing spells postoperatively, and second, in patients who have a low vital capacity, and who have lost a considerable portion of their lung volume. Patients with extensive bilateral disease, and especially those receiving bilateral collapse therapy, more frequently exhibit the tendency to reexpand, unless the pneumothorax pressure can be accurately controlled. More difficult and demanding problems of pneumolysis have been created by the wider application of bilateral pneumothorax, thoracoplasty and contralateral pneumothorax and selective and partial pneumothorax for the remaining lung when the other has been totally destroyed.

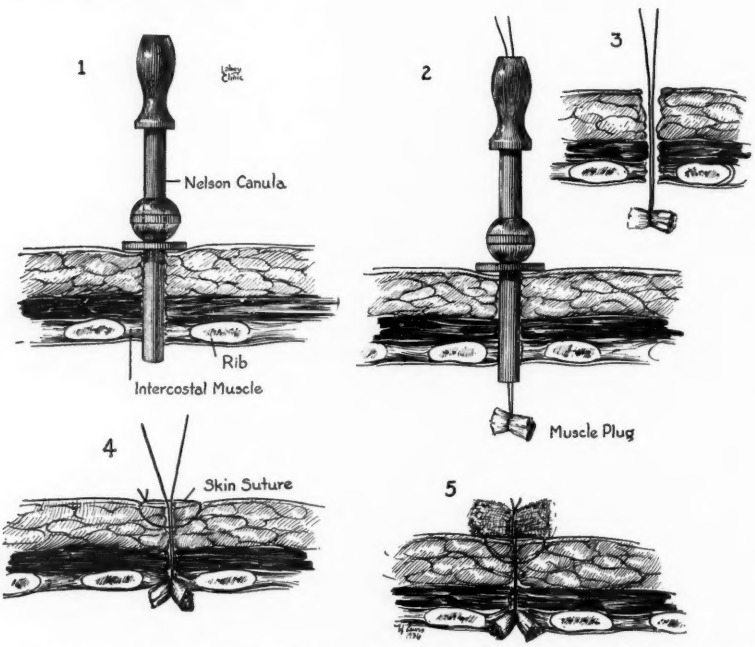


Fig. 2.—Drawings which illustrate the use of a muscle plug in closing trocar wounds of the chest. *1*, cannula in chest wall; *2*, muscle plug inserted. Note double catgut strands emerging through cannula. *3*, Cannula withdrawn; *4*, muscle plug drawn into pleural opening and cutaneous suture inserted; *5*, catgut strands tied over a small roll of gauze.

The great majority of patients can be carried through the postoperative period following pneumolysis without difficulty. Deep sutures, inclusive of all tissue down to the ribs, compression dressings, attention to the postoperative position of the patient in bed and frequent intrapleural readings and refills if necessary all aid in keeping the lung separated from the costal pleura. Regardless of these precautionary measures, a loss of air does, on rare occasions, take place and the result of the pneumolysis is jeopardized or its objective defeated.

The difficulties of closing the interrib and parietal pleura opening by means of suture in the chest wall is obvious. The movements of the cannula, pivoted between two ribs during the course of the operation, enlarges the pleural opening. Approximation of this portion of the trocar wound, no matter what type of suture is employed, is prevented by the presence of the ribs on either side of the interspace. For the same reason, a compression dressing is also ineffective in closing the pleural and intercostal portion of the wound. Variable amounts of air may then escape into the tissue planes of the chest wall and spread laterally without regard to cutaneous sutures or pressure bandages, if a forcible coughing spell raises the intrapleural pressure (Fig. 1).

An experience with one patient who completely lost a very much needed selective collapse led to the use of a muscle plug closure in selected cases. Such a closure is now used when we anticipate difficulties in regulating the pneumothorax postoperatively (Fig. 2). At the completion of the intrapleural procedure, an inch and a half extension is made to one of the trocar incisions and two small sections of muscle are obtained, each approximately $1.5 \times 0.5 \times 0.5$ cm. The muscle bundle is then tied in its center with a strand of catgut, leaving both ends of the catgut fairly long. The muscle is then inserted in the cannula and pushed into the pleural cavity with the catgut ends left emerging from the cannula (Fig. 2). The cannula is withdrawn and gentle traction made on the catgut strands, drawing the muscle plug into the pleural opening. The cutaneous sutures are then inserted, and finally the catgut strands are tied around a small roll of gauze.

An airtight closure makes it possible to regulate accurately the pneumothorax pressure during the postoperative period. A pressure reading is made immediately following the operation and air is withdrawn or injected as indicated. The regular schedule for refills can then be resumed. Emphysema of the tissues of the chest wall has not been noted when this method of closure has been employed. The occlusion of the internal opening with such a plug of tissue serves another useful purpose. The muscle plug will check bleeding in the pleural opening of the wound and prevent its access into the pleural cavity.

TOTAL PNEUMONECTOMY IN PULMONARY TUBERCULOSIS

Report of a Case*

G. E. Lindskog, M.D.
New Haven, Conn.

THE surgical collapse therapy of advanced pulmonary tuberculosis is not uniformly successful, even following an extensive and apparently adequate mechanical collapse of the bony thorax. Two conditions may militate against a good clinical result; namely, (1) rigid cavitation (usually apical); (2) bronchiectatic disease, which may or may not be primarily tuberculous.

Bronchiectasis, when not of tuberculous origin, is treated successfully by radical extirpation of the diseased parenchyma. Various techniques of lobectomy have been perfected through the pioneer work of Graham, Churchill, Heuer, Shenstone, Brunn and others.

When bronchiectasis is related to tuberculosis, there is little experience to guide one in the question of radical extirpation. Forlanini in his classical series of papers (1882) discussed radical ablation of tuberculous lung before considering the revolutionary proposal of artificially induced pneumothorax. He came to the conclusion that radical extirpation was theoretically sound in selected cases, but not practical at that time from the standpoint of technique. Concerning the experimental approach a sporadic report has appeared, chiefly in the Italian literature.

Doyen resected the apical portion of a tuberculous human lung in 1886, and in 1891, Tuffier performed a similar operation. Heuer collected from the literature between 1880 and 1920 nine cases of resection or lobectomy for tuberculosis. Since the latter date, there has appeared a report in this Journal of right upper lobectomy for apical cavitation by Freedlander.

The specters which have haunted the procedure of extirpation in a hopelessly damaged tuberculous lung are (1) hematogenous or bronchiogenic spread of the disease, (2) residual tuberculous empyema requiring subsequent treatment by thoracoplasty, (3) persistent bronchial fistula, (4) high operative mortality for the above and other reasons. Thoracic surgeons have most unanimously avoided extirpation therapy in favor of less hazardous collapse therapy.

In April, 1936, the author unwittingly performed a left total pneumonectomy on a tuberculous bronchiectatic lung, having been led to operation by an erroneous diagnosis. The case report is the subject of this paper.

*From the Department of Surgery, Yale University, The School of Medicine.

CASE REPORT

The patient, E. R., a white sixteen-year-old school girl, was admitted to the Surgical Service of the New Haven Hospital on March 16, 1936, complaining of fever, cough, weakness, and weight loss. Always a delicate child and very susceptible to upper respiratory infection, she was troubled the entire summer prior to admission by cough which was unproductive and easily controlled by cough medicine. In October of 1935, she had a severe respiratory infection.

During the first week of January, 1936, she went to bed one night feeling extremely weak and tired, and was awakened the following morning by a sharp pain in the left scapular region; the pain was aggravated by breathing. In a few days

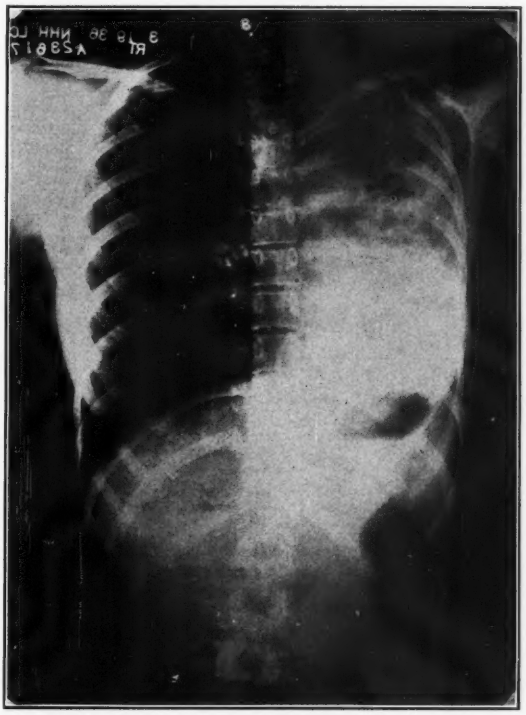


Fig. 1.—Roentgenogram of chest prior to operation (overexposed technique).

she had a very severe cough without much sputum. After three weeks it was decided, without a positive sputum, that she had pulmonary tuberculosis, and she was sent to a state sanatorium.

Her course there was continuously febrile, with a weight loss of 15 pounds, increasing amounts of sputum, complete loss of appetite, and frequent vomiting of recently ingested food. Repeated search of sputa during her sanatorium stay revealed no acid-fast organisms.

There was no immediate family or contact history of tuberculosis. On admission to the hospital the temperature was 102.4°, pulse 120, respirations 32, and blood pressure 112/68. The patient appeared to be a tired, markedly emaciated girl, chronically ill. She was so weak that she was unable to sit up without support. The

left chest was definitely contracted and the respiratory excursion poor. There was dullness to flatness on percussion throughout the entire left chest and widely distributed coarse bubbling râles which occasionally could be heard without the stethoscope. The right lung was clear. There were no cardiac murmurs, and the rhythm was regular. Examination of the abdomen and the extremities was negative; there was neither cyanosis nor clubbing of the nails.

Admission laboratory work revealed a red count of 4.30 million, with 75 per cent hemoglobin; the blood Kahn was negative. The sputum ranged around 20 and 30 gm. daily, was tenacious, mucopurulent, and not foul. Ten successive examinations revealed no tubercle bacilli, and a culture showed hemolytic streptococcus and staphylococcus albus. A Hiss capsule stain for spirochetes revealed only one or two organisms in a fifteen-minute search. The tuberculin test was negative at 1/50 mg., slightly positive at 1/10.

There was a daily fling of temperature to 103° or 104°, an irregularity in the respiratory rate, and a steady weight loss even with postural drainage, transfusion, and high caloric feeding. Stereoradiographic examination of the chest (Fig. 1) revealed an almost complete opacity over the left lower lung field associated with elevation of the diaphragm and displacement of the trachea and mediastinum toward the left chest. There were large cystic areas of rarefaction throughout the left upper lobe.

Bronchoscopic examination ruled out foreign body and tumor. Lipiodol studies revealed a normal right bronchial tree; the left showed extensive saccular dilatations wherever filling occurred.

Complete lung volume studies were carried out according to the technique of Christie with the following results:

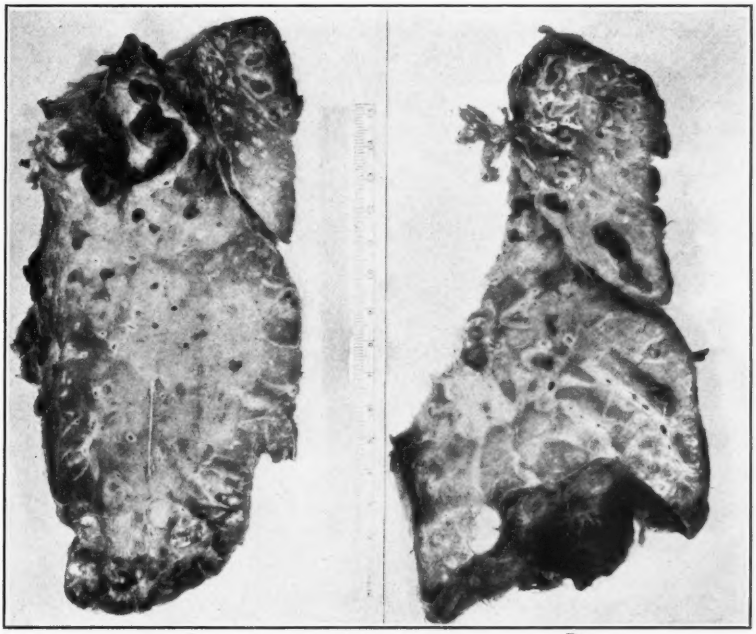
Subtidal	1,200 c.c.
Vital capacity	1,160 c.c.
Reserve	220 c.c.
Tidal	350 c.c.
Complementary	590 c.c.
Total volume	2,140 c.c.

A clinical and roentgen diagnosis of cystic bronchiectasis, atelectasis, and fibrosis involving the entire left lung was made. On April 4, 1936, under novocaine anesthesia, the left phrenic nerve was crushed.

On April 18, 1936, under a basal anesthesia of rectal paraldehyde with intratracheal cyclopropane, an exploratory thoracotomy was performed through a long incision in the fifth left interspace. The lung was diffusely adherent, and several small pleural pockets containing hazy serous fluid were encountered, a bacteriological study of which revealed nonhemolytic streptococcus. By sharp and blunt dissection, the entire lung was freed up from the parietal pleura, the very edematous and infiltrated pulmonary ligament transected and the hilus exposed. Using a tourniquet the lung was amputated, and the hilar stump was closed with individual catgut sutures to the large vessels, a continuous chromic No. 00 suture to the tissues of the stump and an inverting suture of the same material to the pleural surface. The chest was closed with intercostal drainage. The drainage reached a maximum of 750 c.c. on the third postoperative day. It then gradually subsided in amount and became purulent in type.

An oxygen tent was used intermittently until the sixth day. The chest wound showed gross infection and was treated by removing the sutures, and packing with acriflavine gauze following which active healing by granulation occurred. On the tenth postoperative day, there was noted over the right middle lobe area a slight

but definite friction rub which disappeared within twenty-four hours. The patient had a good deal of cough but raised very little sputum. Repeated smears were made of the chest drainage fluid, and on the fourth attempt, one acid-fast organism was found. On May 13, it was clear that a bronchial fistula had been established. The patient began to have a good deal of pain from the intercostal catheter, and on May 16 a thoracotomy with rib resection was done. A large drainage tube flush with the chest was substituted for the intercostal catheter. The daily amount of sputum postoperatively was never enough to record, but specimens obtained on July 4 and July 17 were found to be positive for acid-fast bacilli in scant numbers. Beginning about the first of July, there was a gradual downward trend in temperature and pulse. Because of the bronchial fistula, it was impossible to measure



A. *B.*

Fig. 2*A*.—Longitudinal section showing apical cavitation in lower lobe.
B.—Longitudinal section showing bronchiectatic dilatations at base of the contracted upper lobe.

the actual capacity of the residual empyema cavity, but it was estimated to be about 200 c.c. Due to the necessity for prolonged hospitalization with its financial burden, the patient was transferred to the Norwich State Tuberculosis Sanatorium on the ninetieth day after pneumonectomy. At that time she weighed 70½ pounds. Maximal daily temperatures ranged around 99.8°, the pulse rate was 90, and respirations 22.

Dr. R. Glen Urquhart kindly reported on the patient's status, May 24, 1937 (more than one year later). Her general condition seems about the same, but her mental status is much improved. The chest sinus is still open with rubber tube drainage, and the exudate heavily positive for acid-fast organisms. Recent roentgenograms show very little change in the remaining lung: tuberculous infiltration persists, but

cavitation does not appear. Patient coughs occasionally, especially at night, but raises no sputum. There has been no change in weight. Temperature fluctuates daily between 97° and 99.5°, occasionally touching 101.°

PATHOLOGIST'S REPORT

The gross specimen consisted of a left lung measuring 18 × 10 × 5 cm. in the fixed specimen. The external surface of the lung had a mottled, purple-pink appearance, with numerous fibrous tags particularly around the apex. The upper lobe seemed small, and the interlobar fissure was very poorly defined. On section the apex of the lower lobe revealed a cavity measuring approximately 5 × 4 cm. in its greatest diameters (Fig. 2*A*). At the base of the upper lobe there were several oval-shaped bronchiectatic dilatations containing a yellowish white creamy and soft material (Fig. 2*B*). All of the microscopic sections showed essentially the same picture. There were extensive caseous and ulcerative lesions, many dilated bronchi, and a few areas showing thick-walled alveoli, with or without an organized exudate present in the lumina. The pleural surfaces showed dense hyalinized connective tissue adhesions and some fresh granulations. The mucosal layers of the bronchi were frequently ulcerated and desquamated, and the walls thickened by a marked increase in fibrous tissue. The stroma showed profuse fibroblastic proliferation, areas of caseous necrosis, epithelioid cells, and multinucleated giant cells. Acid-fast stains were done on several sections and tubercle bacilli identified. Pathologic diagnosis: Left lung, showing extensive caseous and ulcerative tuberculosis; tuberculous bronchiectasis.

In Memoriam

EDGAR WARDEN PHILLIPS

1888—1936

Edgar Warden Phillips was born in Bath, N. Y., on October 24, 1888. His early years were spent in the beautiful Finger Lake district of New York State; later, during his busy professional years, many a summer vacation was spent in the old family home on the shores of Lake Keuka. There he could relive with his children the happy days of his boyhood.

Edgar Warden Phillips

Graduating from Cornell Medical School in 1911, Dr. Phillips was appointed interne on the second surgical division at Bellevue Hospital and remained for additional service as resident in surgery. During 1914 he moved to Rochester, N. Y., hardly more than sixty miles from his birthplace. Three years later he joined the Army Medical

Corps and saw active service in France with Evacuation Hospital No. 8 and Base Hospital No. 19. On his return from France, Capt. Phillips was ordered to the Walter Reed General Hospital, Washington, D. C., and assigned to duty on the Empyema Section of the Surgical Service. It was there that the writer met Dr. Phillips and had ample opportunity to learn his many sterling qualities. It was the beginning of a friendship that continued to the time of his death; without doubt, also, the service at Walter Reed was the beginning of Dr. Phillips' career in thoracic surgery.

After his discharge from the army, Dr. Phillips returned to Rochester, N. Y., and resumed the private practice of surgery. Dr. Howard L. Prince, with whom he had become associated, secured for him the privilege of caring for all the empyema cases at the Rochester General Hospital. Dr. Prince states that under Dr. Phillips' skillful care the mortality in these cases dropped from the neighborhood of 35 per cent to 7 per cent, and the period of hospitalization was greatly shortened. It was a natural step from the supervision of empyema cases to the supervision of all thoracic work within the Rochester General Hospital in the capacity of attending surgeon.

In 1926 Dr. Phillips was appointed instructor in surgery at the Medical School of the University of Rochester, thereby becoming one of the very early members of that faculty. Three years later he was promoted to an assistant professorship of surgery and given charge of the teaching of thoracic surgery. Another important position was that of attending surgeon at the Iola Sanatorium (Monroe County Tuberculosis Sanatorium) where a great wealth of clinical material became available to him. He was also consulting surgeon at the Rochester State Hospital.

Dr. Phillips was elected to active membership in the American Association for Thoracic Surgery in 1926. He was loyal in his attendance at meetings and frequently participated in the scientific program. He was on the program for the 1936 annual meeting. His paper is elsewhere posthumously published.

During the summer of 1932 Dr. Phillips suffered a spontaneous perforation of the transverse colon. For months he was desperately sick, but thanks to the unremitting efforts of his devoted friends in Rochester, his life was saved, and eventually it was possible for him to resume an active practice. He was, however, permanently weakened by this catastrophe. A few days before the 1936 meeting of the American Association for Thoracic Surgery he was overwhelmed by bilateral lobar pneumonia and within four days—even while the meeting was in progress in Rochester—Dr. Phillips, still in his prime, died.

Ethan Flagg Butler.

Francis Alexander Carron Scrimger, V.C.
1880—1937

It is with very real sorrow that the American Association for Thoracic Surgery records the death of Dr. Francis Alexander Carron Scrimger, V.C., of Montreal, who died suddenly of coronary thrombosis on February 13 of this year. He was born in Montreal in 1880, the son of the late Rev. John Scrimger, D.D., Principal of the Montreal Presbyterian College. He received his medical education at McGill University, graduating in 1905 with first-class honors in biology. The following three years were spent as interne in medicine and surgery at the Royal Victoria Hospital, following which he did postgraduate work in Europe, principally in Germany, where he studied surgical pathology under Schmorl in Dresden, and surgery under Bier in Berlin. He was then appointed to the staff of the Royal Victoria Hospital in the Department of Surgery. At the outbreak of war he went overseas with the first contingent from Canada as medical officer to the fourteenth battalion. For distinguished bravery, while in charge of a field dressing station at the second battle of Ypres, he was awarded the Victoria Cross. He also served at the Canadian General Hospital No. 1 at Etaples, at the Granville Hospital in England, and was later in charge of No. 3 Canadian General Hospital (McGill Unit) at Boulogne, with the rank of lieutenant colonel. He was on active service for nearly five years, and upon his return to Canada rejoined the staff of the Royal Victoria Hospital. From this time until his death his progress in the Hospital and in the University was one of steady advancement, culminating in the position of chief surgeon of the Hospital in January, 1936, and Associate Professor of Surgery at McGill on the same date.

Dr. Scrimger's fineness of character, his great intellectual capacity, his wide range of surgical knowledge, his originality of mind, and his devotion to his profession, brought him the affection and appreciation of his confreres and associates, and the recognition of scientific bodies both in Canada and abroad. He was a Fellow of the American College of Surgeons, of the American Surgical Association, and of the International Society of Surgery; he was also a member of the Interurban and the Halstead Societies. His published papers embraced both experimental and clinical research. The members of the Association will be particularly interested to know that he made three valuable contributions to thoracic surgery. He was among the first, if not the first, in this country to write upon postoperative massive collapse of the lung, a subject with which he became familiar in the war. Operation for idiopathic dilatation of the esophagus, which he described in two papers, is an original and valuable addition to the difficult treatment of this disease. In the treatment of chronic lung abscess he proposed and carried out with success the implantation of

a pedicled muscle flap into the depths of the cavity, a procedure which was found to insure healing and to shorten by many weeks the period of cure.

Dr. Scrimger's death is a loss not only to his Hospital and his University, but also to surgical science in general. He is mourned by the members of this Society and by confreres throughout the States and in Europe.

Edward Archibald.

Henry S. Plummer*

Dr. Henry S. Plummer was born in Hamilton, Minnesota, on March 3, 1874, the son of Dr. Albert Plummer and Isabelle Plummer. He died in Rochester, Minnesota, on December 31, 1936, of cerebral thrombosis. His early education was obtained in the local public schools and at the University of Minnesota. He attended the Medical School of Northwestern University, from which he received the degree of Doctor of Medicine in 1898. His first three years of practice were spent with his father in Racine, Minnesota, and in 1901 he entered the Mayo Clinic, where he continued the practice of medicine until his death.

Dr. Plummer was known throughout the medical world as a great physician, and his greatest work and interest were in the problems associated with the function and diseases of the thyroid gland. His interests, however, were by no means confined to this field. In the early years of his practice he became greatly interested in the physiology of the thorax, lungs, and heart. He was one of the early investigators of the relationship of negative intrathoracic pressure to respiratory function as well as one of the first to study the use of oxygen as a therapeutic agent. Because of his interest in the fundamental principles underlying diseases of the thorax, he became one of the charter members of this Society in 1918, and was elected to its vice-presidency in 1920. During the earlier years of his practice he was interested in bronchoscopy and esophagoscopy. His early work in the treatment of cardiospasm and esophageal stricture resulted in an important contribution to medicine, and the hydrostatic dilator and esophageal sounds, which were first designed and made by him in his own workshop, are still in use in the treatment of these conditions. Although in later years his interest and most of his contributions became more confined to general medical subjects, particularly those relating to the thyroid gland, he never lost his interest in the principles underlying the treatment of intrathoracic lesions, particularly pulmonary lesions, and those associated with anoxemia.

*Read before the Twentieth Annual Meeting of the American Association for Thoracic Surgery at Saranac Lake, N. Y., May 31, June 1, 2, 1937.

Although Dr. Plummer was a great authority in medicine, he was also considered an authority on literature, architecture, art, music, and horticulture, especially the cultivation of flowers. He was a mechanical and inventive genius. He was a keen observer and had a remarkable ability to correlate the masses of facts which he accumulated. He had great knowledge of human problems and his philosophy of medicine was tempered by a wide humanitarian outlook. His sense of fairness was outstanding. In him were combined to a remarkable degree the finest characteristics of scientist, investigator, and physician.

Stuart W. Harrington.

The American Association for Thoracic Surgery

Officers

PRESIDENT, STUART W. HARRINGTON, ROCHESTER, MINN.
VICE PRESIDENT, HAROLD BRUNN, SAN FRANCISCO, CALIF.
SECRETARY, RICHARD H. MEADE, JR., PHILADELPHIA, PA.
TREASURER, EDWARD D. CHURCHILL, BOSTON, MASS.
EDITOR, EVARTS A. GRAHAM, ST. LOUIS, MO.

Council

LEO ELOESSER, SAN FRANCISCO
FRANK B. BERRY, NEW YORK
EMILE HOLMAN, SAN FRANCISCO
NORMAN BETHUNE, MONTREAL
DAN C. ELKIN, ATLANTA

Twenty-First Annual Meeting of the American Association for Thoracic Surgery

The next annual meeting of the Association will be held in Atlanta, Georgia, on April 4, 5, 6, 1938. The headquarters will be at the Atlanta Biltmore Hotel.

Program Committee

Frank Boland, Atlanta, Ga.
T. C. Davison, Atlanta, Ga.
Dan C. Elkin, Atlanta, Ga.
President, Secretary, Editor, ex-officio.

The closing date for submitting applications for places on the program at the next meeting will be February 1. Those intending to contribute are requested to send comprehensive abstracts of their papers to the Secretary, Dr. Richard H. Meade, Jr., 2116 Pine Street, Philadelphia, as early as possible.

In accordance with the amendment to the By-Laws dealing with the nomination of new members (Art. IV, Sec. 1), the closing date for the year will be December 1, 1937. Members wishing to nominate candidates must secure the proper forms from Dr. Ethan Butler, Chairman of the Membership Committee, Biggs Memorial Hospital, Ithaca, N. Y., and return them to him before that date.